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A COMPREHENSIVE PLAN FOR THE TREATMENT OF THE TUBERCULOSIS PROBLEM.[1]

By Victor G. Bloede,

Chairman of the Executive Committee, Hospital for Consumptives of Maryland (Eudowood), Baltimore, Md.

It is not the purpose of this paper to discuss any new plan for fighting tuberculosis, nor indeed have I such to offer, but my object will be to consider as the only comprehensive and really adequate plan for the treatment of the whole matter the co-ordination, bringing together and working in conjunction under one harmonious management, of the various powerful single factors already so successfully at work in this field.

It is a strange fact that nearly every community which has awakened to the importance of taking measures against this disease has commenced the work by the establishment of sanatoria for incipient cases, while advanced patients, the chief factors in the spread of the disease, and directly or indirectly responsible for the large majority of all our incipient cases, have remained uncared and unprovided for.

In another way this general beginning at the wrong end, so to speak, in giving sanatoria the first consideration while advanced cases are neglected, has deprived the cause of much of its force with the public in that the care of incipient cases, dealing as it does with people who look well and recover, and whose real condition is only apparent to the skilled physician, does not appeal to the sympathy of the masses as those advanced cases in which the extreme illness and suffering of the afflicted person become apparent to the most unobserving and instantly enlists the sympathy and aid of their fellowmen.

This all but universal first attention to the early cases instead of the advanced cases has therefore in my opinion been one of the great drawbacks to the more rapid progress of our work.

Another drawback is to be found in the fact that not sufficient effort has been made to convince the larger public that every community within itself is amply able to finance the factors necessary to battle successfully with this great problem if only its full significance to every home and household were clearly understood. Right here it may be advisable to call attention to the fact that in order to avoid any just criticism and to hasten the progress of the cause, it is absolutely necessary to secure a sound, business-like and economical administration of the funds the public is called upon to furnish and to be able to show clearly that every dollar that is given by the public or state is bearing fruit in the restored health, activity and usefulness of those who are suffering with this terrible malady in its early or curable form, and providing the greatest

[1] A paper read before the International Congress on Tuberculosis, Washington, D. C., September 30, 1908.

possible comfort and happiness to those unfortunates who are marked for death.

It may be proper in these preliminary remarks to refer to the question of the best location for the hospital, sanatorium and farm colony. Not many years ago, it was the general opinion that the successful treatment of consumption required a high altitude, and the result of this was that patients to receive treatment had to go long distances from the centers of population. I believe my honored friend, Dr. Philip of Edinburgh, Scotland, was among the first to combat this theory, and prove the entire efficiency of treatment near home by the establishment of the Victoria Hospital for Consumptives just outside the limits of this classic Scotch city. His opinion, based upon long practical experience is, I believe, that both hospital and sanatorium, and especially the former, should be in close proximity to the great centers of population. Our own distinguished fighter of the White Plague, Dr. Flick of Philadelphia, holds the same view, and claims that in his hospital in the very center of the busy city of Philadelphia, he can effect practically as many cures of curable cases as the average institutions far from the din of the great cities.

My own experience and study confirm this view. While no doubt the air at high altitudes is more grateful and healing to the lungs of the sufferers and the low temperature, especially in mid summer, may be conducive to more rapid recovery, it is nevertheless a fact that the best kind of work can be done right at home, in some quiet place just outside of our great centers of population.

One of the explanations for this I think lies in the mental attitude of the patient. Near home, and within easy reach of those dear to him, the pangs of homesickness are practically unknown, the patient's mind is at ease, and he gives himself up wholly to the treatment without experiencing the gnawing and destructive mental suffering to which the large majority of patients who are removed far from home and friends are subject. The mere feeling that they are within thirty minutes ride of their friends and family and can communicate with them at any moment, in my opinion begets a mental quiet more favorable to and potent in effecting a cure than the finest mountain air with a spirit of melancholy gnawing within.

I have already stated my proposition, namely, that to fight tuberculosis adequately, neither sanatorium nor hospital working as independent forces will prove most widely effective, and I will now proceed to the consideration of a plan which my experience of several years in this work leads me to believe would prove the most effective system that can be devised to secure rapid and permanent results.

This plan contemplates the combined and simultaneous operation of the four great agencies, each one indispensable within itself; viz.:

First, the dispensary.
Second, the hospital for advanced cases.
Third, the sanatorium.
Fourth, the farm colony.

All of these four great forces to be co-operating, and each one forming an integral part of one great system. I believe where such a combination can be made (and I see no reason why it should not be feasible everywhere), the end of the great white plague is within sight, at least of the coming generation.

In order to make myself clearly understood, I desire to briefly define the co-relation and co-ordination of all of these single agencies as I understand them, but before entering upon this subject, I wish to say a few words upon a subject about which we are all so often asked and that is, the proper organization for carrying out this work in any of our communities.

I should say that the first requisite is the awakening of the public in any given community to the importance of this work, not in the abstract, but in its bearing upon every home. There is no better or quicker method of accomplishing this than through the press, and I know from our own experience in this work that the power of the press is the greatest and most potent single element available for stimulating the public conscience and educating and awakening the public to the importance of the subject. I believe that to the work of the press of Baltimore, unselfishly and freely given at all times, a large part of our success, as well as financial support (contributed by all classes of our community), is due.

The church and church organizations should also be a great factor in this work, but for some reason or other this great influence has not yet taken its proper place in our work or made its true power felt in the way of practical results either by adequate co-operation in its propaganda, or financial aid secured through its appeals.

The next most important step to be taken in the line of organization is the selection of progressive men and women to direct the work, men and women who are workers and not too busy to give some time and thought to the cause of their less fortunate fellowmen.

These should be selected from different stages of our social order and from all walks in life, from the church, from professional, business and political life, from labor circles, in short wherever true interest is shown in our work, for humanity should know no caste. Next you should select from among this varied material a competent and aggressive managing committee. This committee should be made up of men possessing executive ability, who have had a good business training, and who command the confidence of the public. Last, but not least, a medical director must be found willing to put his best life's blood into this work.

Such men are not easy to find, but every large community has such material in its midst, and with the proper public support, the progress of the work will be rapid and gratifying. The limits of this article will not permit my going into further details, and I therefore have to content myself with a merely cursory reference to this most important subject of proper organization, upon which much of the success

of the movement must necessarily depend. Before leaving this subject, however, I desire to emphasize the fact that it *is not desirable to seek aid from state or municipality at too early a stage of the work* because even if a community is successful in obtaining it, the amount given is usually small and inadequate while the general public as a rule assume that when the state or city takes up a given problem the responsibility of the individual ceases, and as a consequence, their interest in the work lags, and the contributions from private sources dwindle.

After the proper organization has been effected on the lines indicated, the first important step is the establishment of

THE DISPENSARY.

To this dispensary all suspicious cases should be sent for investigation, diagnosis and classification. The dispensary has been fittingly called the clearing house for tuberculosis cases, and is the surest means the community can employ to promptly and accurately learn the condition of its sick, and to accomplish the greatest possible amount of good for the least expenditure of funds, as through it, the great majority of the cases in any community can be reached and instructions and help be given. In the larger cities this dispensary might form part of some general dispensary or hospital. In Baltimore, our institution has depended almost entirely upon the Phipps Dispensary of the Johns Hopkins Hospital for the diagnosis and classification of its cases, although we invite and receive the co-operation of other hospitals and dispensaries of the city. All applications received for admission to our hospital are first referred to the dispensary where a thorough examination and diagnosis of the applicant are made, our resident physician being one of the examiners, and upon this report our admission or rejection of the patients is based.

Those who have been active in this work know how difficult it is sometimes made for those in charge to reject patients whose admission is insisted upon by strong political or social influences. The applicant may for various reasons be unfit for admission, yet it may be a very difficult task for the board of directors to reject him. By requiring the certification of all patients by the dispensary physicians as an essential step, this trouble is wholly overcome, and when it is once understood that all social or political influences in the absence of such certification are disregarded in the admission of patients, a very unpleasant duty and responsibility is lifted from the shoulders of the directors. The dispensary of course should be in charge of some active physician who has made a specialty of the diagnosis and treatment of tuberculosis, and who has had the widest possible experience.

To be productive of the highest good, the dispensary should also be provided with visiting nurses who report upon the home condition of the patients in such cases where home treatment is considered sufficient, see that the instructions of the doctors are carried out, and persuade those who are exposed to possible infection to come to the dispensary for observation, diagnosis and instructions. It is not always necessary in the beginning to have paid nurses, as the work of the visiting nurse may be done with a fair amount of success by earnest volunteer workers drawn from the church or society. As the work grows, the medical director will have no difficulty in securing assistance from doctors interested in tuberculosis and his volunteer staff will generally be large enough to meet all requirements.

Those who desire a fuller and more detailed knowledge of the great work that can be done by this clearing house for the whole system (and which constitutes its first element) I would refer to Dr. Louis Hamman, the able and devoted head of the Phipps Dispensary, an institution which I believe, considering the small amount of money expended in its maintenance, produces as great results as are achieved anywhere in the world.

Our next consideration is

THE HOSPITAL FOR ADVANCED CASES.

Of all the single elements forming the system I suggest I consider the hospital for advanced cases the first in importance, for there is no argument which can gainsay its absolute necessity. It seems simply incomprehensible that a great state like Maryland, and many others, up to the present time have provided no place for the care and treatment of advanced cases of tuberculosis, or even a place for the hopeless invalid to die. Indeed up to last year we had only one sanatorium in the entire state, and that accommodating only about 30 patients for the 10,000 or more that are afflicted; less than the proverbial "Drop in the bucket."

Advanced cases being, as experience has shown us, the chief means of propagating and spreading the disease, should therefore in every instance be first provided for. This is the practical view of the situation, but in addition to this from the humanitarian standpoint, to leave advanced cases in their homes, not only to spread infection broadcast, but to gradually approach death after months or even years of worse than death, with improper food and inadequate care, is such fiendish barbarism as to make those of us who know these conditions wonder whether civilization is more than a name. In my opinion therefore, if conditions are such that only one of the various elements necessary to properly battle with tuberculosis can be employed in the community, the hospital for advanced cases should receive first attention. The site selected for such a hospital should be as near the center of population as possible, convenient to railroad or trolley, with a pleasant view, plenty of shade and good water.

It has been deemed advisable by some to locate such advanced-case hospitals in the center of cities, but in view of the fact that land can always be obtained in the suburbs, with freedom from noise and dust, and within easy reach, I see no possible advantage in such a plan, especially in view of the fact that it is now generally conceded that advanced cases should be treated as though they had every chance of recovery, and such treatment can certainly be best given in the country.

The type of building most efficient and economical is, in my opinion, one combining small wards with individual rooms

for the isolation of dying cases or those with disturbing or distressing symptoms such as persistent night coughs. A building of this type, which I think will be found very efficient, has just been erected near Baltimore, and I refer those interested in a further study of this subject to this design as it is the result of considerable study and experience, and contains some novel features. The hospital referred to has a central administration building of two stories with assembly hall, office, dining room and kitchen on the first floor, and staff quarters on the second floor. On each side are one story wings containing four wards of six beds, six isolation rooms, lockers, bathrooms, and wide piazzas upon which the beds can be wheeled. In the basement of the main building in addition to the necessary heating apparatus is a well equipped autopsy room, for pathological work. A laboratory for sputum and other examinations and research work is also to be provided for later.

A hospital for advanced cases built upon these lines gives the greatest efficiency in the treatment of these cases at a minimum cost both as to expense of construction and subsequent maintenance. Our plan also contemplates the construction of shacks and cottages later on for the accommodation of those advanced cases which have improved sufficiently during their hospital treatment to become ambulatory.

The next link in the chain is

The Sanatorium.

In our plan the sanatorium for incipient cases is built upon ground immediately adjoining the hospital for advanced cases, but at a distance of about one quarter of a mile so that while the two institutions are entirely separate so far as the communication of the patients is concerned (which, as you all know, is very desirable) the entire system is under one management. This insures the greatest possible economy in the control and maintenance of both institutions, the necessary supplies being furnished through one central station and the same resident physician having the management and control of both, thus avoiding unnecessary duplication of working forces.

In this country, as you all know, with few exceptions the cottage or shack system for sanatoria has been adopted and found most suitable.

The buildings may be of comparatively inexpensive construction, but expense should not be spared in making the patient's surroundings not only comfortable and hygienic, but as homelike as possible, for the tuberculosis patient, unlike the patient in a general hospital, spends months under our care, and hence the more comfortable and attractive we can make his surroundings, the more rapid and thorough will be his progress and cure. If it is true that the chief value of the sanatoria is educational, then the nearer it approaches the ideal home condition, the greater, more impressive and lasting will be its teaching.

We have found in our work where there is a blending of all classes (as must inevitably be the case in such institutions) that the smaller the groups of patients the easier and more successful will be their administration, for society even under such circumstances where the "touch of nature" should "make all mankind kin" is bound eventually like water to find its own level, and hence the administration of large and heterogeneous groups of patients in close quarters becomes impracticable and undesirable. My limited space will not permit me to go into further details here as to the character and arrangements of the building we have found most desirable and satisfactory in our work in Maryland, but these will be gladly given to those interested.

Before leaving the subject of sanatoria, I wish to speak of one phase of this great problem which has given me much food for thought and study, and that is the question of occupation, mental and physical, for the patients during their enforced idleness of weeks or months while undergoing the cure.

I cannot imagine a more demoralizing influence than taking men and women whose days have been filled with useful work and breadwinning and condemning them to a life of idleness with absolutely nothing else to do than sunning themselves in easy chairs and dwelling upon their own physical ills and frailties. Work and useful occupation of all kinds is the greatest blessing next to health that can be vouchsafed a normal being in this earthly life of ours. Its spiritual as well as therapeutic value to every human being cannot be over estimated. A healthy mind craves for it and finds relief from much bodily and mental suffering through its benign influence. Those who would be content in a life of absolute idleness are fortunately few, and even these few would be better in every way if compelled to do something useful within their strength. I think we are making a very great mistake in not giving this all vital question at least as much care and consideration in the conduct of our sanatoria as the purely physical condition of our patients.

There is no truer saying than "Satan finds some mischief still for idle hands to do," for even if the adage is not taken literally idleness will turn the mind of the patient irresistibly upon his own ills and suffering and he will inevitably dwell upon and nurse these until the whole great world seems to him to center in his case. I cannot help but feel therefore that the average sanatorium as now conducted is a great school for the teaching of indolence to those who can least afford to practice it if indeed any of us can, and I urge upon my hearers to give this phase of our great problem the serious consideration its vital importance to the welfare of humanity warrants, for I believe that up to this time this feature of the problem has been sadly neglected.

The foregoing remarks logically bring us to the consideration of our final agency in this work.

The Farm Colony.

Fraught as this whole tuberculosis question is with great and grave problems there is none greater than the after-care of the arrested cases, for we must all admit that simply prolonging life in those whom we can never hope to restore

again to the world of workers, or make fit to earn their living, is to say the least so doubtful a charity that many truly humane and generously disposed persons have questioned its desirability.

It does perhaps seem a strange paradox that we gently put a hopelessly sick or crippled animal out of its misery on the score of humanity, and yet are bent on prolonging to the utmost the useless suffering of our hopeless sick. Yet such are the ethics of our age, and this is the condition we have to deal with, not as a theory but a concrete fact. We must therefore deem of the highest importance the after-care of the hundreds of arrested cases, men and women who, although temporarily endowed with a further lease of life through the ministrations of our hospitals and sanatoria, are not able through their physical disabilities to return to their accustomed avocations.

For such cases the farm colony must be provided where through light work in field and garden these patients can contribute their mite towards their own support as well as that of the patients in both hospital and sanatorium. Not only does this reduce the cost of the general maintenance of all of these institutions, but it restores the patient to the ranks of self respecting workers, thereby lessening their cares and sorrows as well as physical ills. In this great work we have scarcely passed the initial or experimental stage, and yet we already see great therapeutic, spiritual, and material benefits resulting. We have patients at work a few hours each day on our farm colony who never expected to leave their beds again, and largely as a result of their labor our cost of maintenance per diem in this chain of institutions—and our patients live well—has fallen far below that of any similar institution in our country, of which we have the records.

The farm colony should be located in immediate proximity to the hospital and sanatorium so as to bring it under the same general management. To carry out its purpose it must of course have a large tract of arable land at its disposal for raising hay and other fodder, fruits, vegetables, flowers and garden stuff. On this farm colony should be located a dairy of sufficient size to furnish all of the milk required by these various institutions, and chickens and poultry raising adds another excellent and profitable branch of industry within the scope of the labor available.

In a farm colony of this kind once fairly established and in full working order, sufficient work can be done to make any number of patients practically self supporting, indeed I believe that it may be possible under the most favorable conditions, and by the addition of some other light industries to make the care of the patients return some profit, at least enough to render the payment to them of small wages a possibility.

This whole comprehensive plan for the treatment of the tuberculosis problem, I am happy to say, is not entirely a theory or yet a Utopian dream, but to a large extent an accomplished fact.

It is perhaps too soon to say what the future of our ambitious plan may be or whether or not we will be able to fully encompass the high ideals we have set ourselves, but when I tell you that we have each and every one of those integral parts of the proposed system in co-ordination and harmonious work under one management in our State of Maryland, and that this has been accomplished mainly within the last three years by the aid of an awakened public, competent managers and a resident physician, Dr. Alexius M. Forster, who has consecrated his life to its success, you will grant me that this "comprehensive plan" I have outlined is fraught with great possibilities for suffering humanity.

NOTE.—Since this article was written my attention has been called to the fact that the proposed combination of forces has been in successful operation at the Victoria Hospital, Edinburgh, for some time past, and that the credit for the first practical application, if not origination, of such comprehensive treatment of the problem belongs to Dr. R. W. Philip.

THE AXILLARY DIASTOLIC MURMUR IN AORTIC INSUFFICIENCY.

By RUFUS COLE, M. D.,
Associate in Medicine, The Johns Hopkins University and Hospital,
AND
ARTHUR BOND CECIL, A. B.

The exact situation at which the basal diastolic murmur of aortic insufficiency is usually heard with its greatest intensity has caused much discussion among observers. The area over which it is transmitted, and the situations where it is heard with increased intensity have been less discussed. The following paper deals mainly with these latter questions.

The area of maximum intensity has been described by various observers as being at the aortic area, over the sternum itself, along the left sternal margin, or even at the ensiform. From these conflicting views it is evident that the point of maximum intensity is not constant, but varies under different conditions. Most authorities, however, agree in considering that the murmur is usually best heard to the left of the sternum, in the third or fourth interspace at the sternal margin. Most writers state that it is transmitted downward along the sternum towards the ensiform; a few mention that it may be transmitted to the apex, occasionally heard in the axilla. Osler in the last edition of his text-book states that "in some cases it is loudly transmitted to the axilla at the level of the fourth interspace, not by way of the apex." In Osler's Modern Medicine (Philadelphia and New York, 1908), in the article on Diseases of the Circulatory System,

by Osler and Gibson, note is made of the variability of the point of maximum intensity, but no mention is made of its transmission to the apex or into the axilla. Babcock states that "this murmur is transmitted downward toward the ensiform appendix, and in some instances also toward the left, even as far as the apex."

Much attention has been given to the apical murmur in aortic insufficiency, which is presystolic in time, and has the characteristics of the mitral stenotic murmur, and so differs materially from the blowing murmur of aortic insufficiency. This murmur was first described by Flint, and has since been called the Flint murmur. Interest in it has drawn attention away from another fact, namely, that the blowing, aortic diastolic murmur may be transmitted to the apex or even to the axilla. Foster (Clinical Medicine, Philadelphia, 1874) early recognized that at times the murmur of aortic insufficiency might be heard distinctly at the apex, "equally, if not more so than over the ensiform." He observed this in several cases, in which post-mortem examination showed a lesion of the posterior cups of the aortic valve, and from this he concluded that the transmission of the aortic murmur to the apex in certain cases, "depends on the regurgitation taking place through incompetency of the posterior aortic segment, either at its right angle, or through perforation of its curtain."

He thought that this transmission of the murmur to the apex not only enabled one to decide as to the exact nature of the valvular involvement, but also was of prognostic significance. The posterior segment having no coronary vessel opening above it, a lesion involving this segment alone would not have so much effect in disturbing the coronary circulation as though the lesion involved the other segments, above which the coronary vessels open. Both theoretically and from his observation of cases, therefore, he concluded that the prognosis was less serious where the murmur was transmitted to the apex. Very little attention has been given to his observations, and his conclusions, as regards prognosis at least, have not been confirmed. It is altogether probable that the fact that he began to base theories upon observations, which in themselves were correct, caused the observations themselves to be disregarded and discredited. He distinctly states that the murmur he described "though diastolic in time, differed from a diastolic mitral murmur *in diminishing instead of increasing in intensity up to the first sound.* This character enabled me at the time to diagnose it as an aortic diastolic murmur transmitted to the apex; and to disregard the hypothesis, that it was due to mitral stenosis." It is, therefore, quite evident that he was not discussing the rumbling, presystolic murmur at the apex previously described by Flint.

Opinions differ very greatly in regard to the frequency with which the Flint murmur is present in cases of aortic insufficiency. Many of the German observers doubt its occurrence, and so good an observer as Gibson states in his text-book that he has never yet been able to demonstrate it. On the other hand it is being recognized frequently by the staff of the Johns Hopkins Hospital, and Thayer,[1] in a study of fatal cases of aortic insufficiency, states that in 74 cases coming to necropsy at the Johns Hopkins Hospital, a murmur of mitral stenosis was heard at the apex some time during the observation of the patient in 45, or 60.8 per cent of the cases. In 33 of these cases no stenosis of the mitral valve could be demonstrated. We ourselves are quite familiar with the Flint murmur, and the one we are describing is quite distinct from this murmur (or that of a true mitral stenosis).

The Flint murmur has all the characteristics of that occurring in true mitral stenosis, being rumbling and echoing usually presystolic in time, limited to the region of the apex, and usually ending in a sharp first sound, and so differing from the prolonged, blowing diastolic murmur heard in aortic insufficiency.

The attention of one of us was directed to the fact that the murmur of aortic insufficiency might be distributed widely throughout the axilla, when on several occasions in the hospital wards, notes were made on the histories of cases of aortic insufficiency that "there is present at the apex a prolonged, blowing systolic murmur transmitted into the axilla;" but upon accurate study these murmurs were found to be not systolic, but diastolic in time, and of exactly the same character as the diastolic murmur heard at the base. These murmurs, however, were not directly transmitted from the base, that is, they were not heard less and less distinctly as one passed outward from the point of maximum intensity into the axilla, but they were heard quite loudly outside of the apex, grew fainter as one passed inward, and then increased in intensity as the sternum was approached. Afterwards one of us had a patient under observation, in whom there was heard, on admission to the hospital, only a soft, prolonged diastolic murmur at the apex and in the axilla, while at the base and over the body of the heart no murmurs were audible. Later, following rest in bed, there developed a typical aortic diastolic murmur along the left margin of the sternum. All the other features of aortic insufficiency were present, and in this case a diagnosis of aortic insufficiency was made, notwithstanding the peculiarity of the aortic murmur on admission. In another patient the murmur in the axilla remained constant, while during the period of observation no murmur ever appeared in the usual situation. In this case also, the other features made the diagnosis of aortic insufficiency necessary. Thus having our attention drawn to the localization of an aortic diastolic murmur in the axilla we began to pay special attention to the occurrence of this murmur, and decided to make an accurate study of the distribution of the diastolic murmur in a number of cases of aortic insufficiency.

The cases studied have all shown well-marked features of insufficiency of the aortic valves. The method of study has been to place the patient in an upright position, or, if confined to bed, to place him in a semi-recumbent posture, as near upright as possible. The area of deep cardiac dulness has been outlined by percussion, and the boundaries have been marked with a pencil on the chest wall. Then the area over which the aortic diastolic murmur has been heard with greatest intensity

[1] Am. J. M. Sc. Phila., 1901, CXXII, 538.

has been indicated by cross lines, and the area over which it is transmitted has been carefully determined by passing outward from this point of maximum intensity in all directions, and the outermost limits, where the murmur is just audible, have been indicated by dotted lines. Where there is an area of secondary intensity in the axilla, this also has been indicated by cross lines. Photographs were then taken of a number of these patients showing the areas above mentioned. We have found diagrams very unsatisfactory for such purposes, as chest walls differ so much in size and shape, and diagrams and measurements alone give a very imperfect idea of the conditions present.

CASE I.—J. G. Disp. No. 22,204 E. Black; age, 64; sailor.

Diagnosis.—Aortic insufficiency, dilatation (aneurysm?) of thoracic aorta, arterio-sclerosis, emphysema, chronic nephritis.

This patient was admitted complaining of shortness of breath and indigestion.

He has never had acute rheumatic fever. He has been a regular, but moderate, drinker, usually taking gin.

His main symptom has been shortness of breath, which is increased with muscular exertion. This interferes with his work, and it is for this reason that he comes to the hospital. He has also had some distress after eating. He has had no pain in his chest, and no acute symptoms of broken compensation.

On examination he is found to be a moderately well-nourished man. There is a marked grade of arterio-sclerosis. The pulse is collapsing, vessels throbbing.

Examination of Heart.—The point of maximum impulse is in the VI interspace, 2.5 cm. outside the mammillary line. It is sharp and punctate. There is a well-marked pulsation in the I and II left interspaces in the parasternal line. The first left interspace shows some fullness as compared with the right.

On palpation at the base there is felt a quite well-marked diastolic shock, most marked in the II left interspace. The impulse at the apex is heaving; no thrill is felt.

Dulness extends 15 cm. to the left in the V interspace, 4.5 cm. to the right in the IV interspace. There is a definite impairment of resonance over the sternum, as shown in the diagram.

On auscultation at the apex both sounds are well heard. The first sound is rather loud and is clear at present. The second sound is louder than the first. At the point of maximum impulse, and in a limited area just about it and below it, the area measuring not more than 4 cm. in diameter, there is heard a fairly well-marked rumbling murmur, presystolic in time, crescendo, ending in the first sound, therefore having the characters of the mitral diastolic murmur, and probably being a Flint murmur. But, in addition to this, there is heard a well-marked prolonged diastolic blowing murmur, quite distinct and different from the presystolic rumble previously mentioned. This has a much wider distribution than the rumbling murmur, the area over which it is audible being indicated by the dotted line in the diagram. In the VI interspace, 5 cm. outside the point of maximum impulse, the murmur is heard quite loudly, and it is heard with diminishing intensity as one passes away from this point in all directions. The murmur is prolonged, extending throughout the entire diastole, is blowing in character, and has all the characteristics of an aortic diastolic murmur. It may be traced inward, and is found to be continuous and identical with the aortic diastolic murmur heard at the base. As one passes inward, however, the murmur becomes fainter, and in the nipple line is just audible, but its intensity increases as one approaches the sternum. It is heard loudly at the sternal junction of the fourth left rib, but with greatest intensity in the II right interspace, where it is very loud and booming. Passing outward from this point in all directions the murmur diminishes in intensity.

This case shows an aortic diastolic murmur, loudest in the II right interspace, heard as high as the clavicles, as low as the ensiform, over the body of the heart, and in the lower axilla. It is of special interest that in the VI interspace, 5 cm. outside the point of maximum impulse, this murmur is heard quite loudly, louder than it is immediately inside of this point. There is thus an area of transmission of the murmur from the point of greatest intensity, and a second area of transmission of the murmur from the point of secondary intensity in the axilla. This case also shows a fairly well-marked Flint murmur.

CASE II.—C. T. Gen. No. 63,459. Med. No. 22,485. Black; age, 45; occupation, porter.

Diagnosis.—Aortic and mitral insufficiency.

This patient was admitted complaining of shortness of breath and dropsy.

He has been a very hard worker, and has a marked alcoholic history. He has had no acute illnesses, no acute rheumatic fever or chorea.

The symptoms of which he now complains first appeared one year ago, and followed exposure. At present there is considerable general œdema. His vessels are quite markedly sclerosed. The pulse has a collapsing character.

Examination of Heart.—The point of maximum impulse is in the VI interspace, 13 cm. from midsternal line. The impulse is wavy. No thrill is felt at apex or base.

Cardiac dulness extends 13.5 cm. to the right in the IV interspace, to the left as shown in the diagram. On auscultation at the apex the first sound is faint, is almost entirely replaced by a rough scratching murmur. The entire diastole is occupied by a rough murmur, slightly rumbling at the apex, but not crescendo. This murmur is heard over the entire left front of chest, but is more blowing everywhere than it is just at the apex. The murmur is heard with maximum intensity in the II and III left interspaces close to the sternum. Here it is very loud, harsh and prolonged. It is loudly transmitted downward along the left margin of the sternum. Passing outward toward the nipple, however, the murmur becomes fainter and then again increases in intensity until one reaches a point in the V interspace about 3 cm. outside the mammillary line. Here the murmur is again heard with marked intensity. It is a question whether the intensity of the murmur is not greater here than anywhere else, even greater than in the III interspace at the sternal margin. The murmur here has not the rumbling character heard at the apex. It is transmitted throughout the entire axilla, the murmur diminishing in all directions as one passes away from the point above mentioned.

This case shows an aortic insufficiency murmur widely transmitted, showing two areas of great intensity, the first in the III interspace at the sternal margin, and the second in the V interspace, 3-5 cm. outside the nipple line. At these two points the murmur is of almost equal intensity. At the apex the murmur has a more rumbling character, but it is questionable whether one should speak of this as a true Flint murmur.

CASE III.—R. D. Gen. No. 63,244. Med. No. 22,419. White; age 16; occupation, laborer.

Diagnosis.—Acute rheumatic fever, aortic and mitral insufficiency, mitral stenosis.

The patient gives a history of recurring attacks of acute rheumatic fever since five years of age. He has had some shortness

of breath on exertion ever since the first attack. At the time of examination he is recovering from an acute attack of rheumatic fever, and the temperature is still elevated. There has been no pain in the joints for the past six days. He is still anæmic and looks rather ill, but is comfortable at present. The pulse shows a well-marked collapsing character.

Examination of Heart.—The point of maximum impulse is in the V interspace, 8 cm. from midsternal line.

The impulse is forcible, rather heaving, not punctate. There is no thrill, no diastolic shock at the apex or base.

The cardiac dulness extends 10 cm. to the left in the V interspace, 5 cm. to the right in the IV interspace, and above to the third rib.

On auscultation at the apex the first sound is replaced by a blowing systolic murmur of moderate intensity, which is transmitted outward into the mid-axilla. There is also heard a rumbling murmur extending throughout the entire diastole. The second sound is not audible. Passing inward, one begins to hear a blowing diastolic murmur, the rough rumbling diastolic murmur becoming inaudible. The blowing murmur increases in intensity as one passes inward until the point of maximum intensity is reached over the midsternum at the level of the third rib and III interspace. The murmur is also audible over the entire right front of the chest, being quite loud at the right sternal margin in the I and II interspaces. The murmur is heard loudly as one passes outward along the upper margin of the heart as far as the parasternal line, but outside this becomes fainter until one reaches the anterior axillary line, 4 cm. outside the nipple. Here the blowing diastolic murmur is again heard with great intensity, almost as loud, if not quite as loud, as in the area of maximum intensity above noted. It quickly becomes faint and disappears as one passes outward and downward. In the axilla it has none of the rumbling character which characterizes the diastolic murmur heard about the nipple over the area shown by the continuous line in the photograph.

This case shows a very widely distributed murmur of aortic insufficiency with the area of maximum intensity over the sternum, but with a second area of great intensity 4 cm. outside the nipple. The murmur at and inside the point of maximum impulse has a more rumbling character, but is prolonged throughout the entire diastole, is not crescendo, and does not end in a sharp first sound, and there is no thrill. It has exactly the time of the aortic diastolic murmur, and it is questionable whether the rumbling character is due to a narrowing of the mitral orifice.

Case IV.—M. P. Gen. No. 66,628. Med. No. 22,547. Disp. No. 22,421 E. White; age, 43; occupation, clothes presser.

Diagnosis.—Aortic and mitral insufficiency, mitral stenosis.

This patient has not been a very hard worker, and has taken alcohol only in moderation. There is no history of syphilis or gonorrhœa. He has had some indefinite joint pains, but no attacks of acute rheumatic fever.

Five months before admission he began to suffer from weakness and shortness of breath on exertion. He has had at times some precordial pain, and he has had some cough.

On examination he is found to be a moderately well-nourished man, very neurotic, and having some shortness of breath, but the symptoms of broken compensation at present are not marked. The vessels are sclerosed and thickened. The pulse is collapsing.

Examination of Heart.—The point of maximum impulse is in the VI interspace, 12.5 cm. from the mid-sternal line.

On auscultation at the apex there is heard a loud systolic murmur well transmitted to the mid-axilla. At the apex, and over a small area just about it, there is heard a well-marked presystolic murmur having the characteristic features of the murmur of mitral stenosis. Over the body of the heart there is heard a prolonged blowing diastolic murmur heard with maximum intensity along the left sternal margin. The murmur is transmitted outward into the axilla over the area indicated by dotted lines in the diagram. Passing outward from the sternum, however, the murmur becomes fainter and fainter as one passes into the axilla, and nowhere about the apex or in the axilla is there a point where the murmur is heard with greater intensity.

This case shows the aortic diastolic murmur transmitted to the axilla with no point of greater intensity outside the mammillary line. The other features of the case renders it probable that the presystolic murmur heard at the apex is due to a true narrowing of the mitral orifice and is not a Flint murmur.

Case V.—J. F. Gen. No. 64,395. Med. No. 22,808. Black; age, 38; occupation, laborer.

Diagnosis.—Aortic and mitral insufficiency.

This patient gives a history of acute rheumatic fever nine years ago, the illness lasting four and a half months. He had gonorrhœa eight years ago; no history of syphilis. There is a history of some alcoholic excess, and he has been a very hard worker.

On examination he is found to be a very muscular, well-developed man. The vessels are moderately thickened, the pulse is collapsing.

Examination of Heart.—The point of maximum impulse is in the VI interspace, 14 cm. from the mid-sternal line.

No thrill is felt at the apex.

On auscultation at the apex there is heard a loud, musical, systolic murmur well transmitted into the axilla. At the apex and over a small area surrounding it there is heard a short, rumbling presystolic murmur ending in the first sound. At the base there is heard a very loud blowing diastolic murmur heard with greatest intensity at the sterno-articulation of the third left rib. It is quite widely transmitted, being audible over the entire area marked by the dotted line in the photograph, and, as indicated, it is well heard in the axilla, but is not audible just at the point of maximum impulse. The murmur in the axilla is directly transmitted outward from the base with diminishing intensity, and there is no area in the nipple over which the murmur is heard with less intensity, and there is no point in the axilla over which the murmur is heard with greater intensity.

In this case the murmur of aortic insufficiency is quite widely transmitted, being audible in the left axilla, but the murmur here is transmitted directly outward from the base. The other features of the case render it probable that in this case the presystolic murmur at the apex is due to a true mitral stenosis, but the absence of thrill and of a tapping first sound, as well as the character of the pulse, make it possible that this is merely a Flint murmur.

Case VI.—J. B. Gen. No. 63,724. Med. No. 22,578. White; age, 28; occupation, carpenter.

Diagnosis.—Aortic and mitral insufficiency, mitral stenosis.

This patient gives no history of acute rheumatic fever or chorea. He has had no acute illnesses. He had a venereal sore 15 years ago, but no history of secondaries can be obtained. He gives a marked alcoholic history.

At present this patient has no marked symptoms of broken compensation. He has shortness of breath on exertion. There is slight sclerosis of the radial artery.

Examination of Heart.—The point of maximum impulse is in the VI interspace, 14 cm. from the mid-sternal line. The dul-

ness extends 15 cm. to the left in the VI interspace and 4.5 cm. to the right in the IV interspace.

The impulse at the apex is rather diffuse and heaving. There is no definite thrill.

On auscultation at the point of maximum impulse, accompanying the first sound is a soft blowing murmur, and preceding it is a loud rumbling crescendo murmur. This is heard only in the region of the apex and just below it. The systolic murmur is transmitted as far outward as the mid-axilla. Passing inward the rumbling presystolic murmur becomes fainter, but one begins to hear a prolonged blowing murmur extending throughout the diastole, increasing in intensity as one approaches the sternum. It is heard loudly over the sternum, and reaches its maximum intensity in the II right interspace. It is heard faintly upward to the clavicles, as shown by the dotted line in the photograph. Passing outward to the left the murmur becomes fainter and fainter, and then increases in intensity until a point in the axilla is reached in the VI interspace, 3.5-5 cm. outside the point of maximum impulse. Here the murmur is again heard with great intensity, though not quite so loudly as it is in the II right interspace. The murmur becomes fainter as one passes in all directions from this point of great intensity in the axilla. This murmur, however, is directly continuous with the basal diastolic murmur, has exactly the same character, and occupies exactly the same time in the cardiac cycle.

This case shows very well the characteristic features of the axillary aortic diastolic murmur, which is quite distinct from the presystolic rumbling mitral murmur heard at the apex. The other features render it probable that in this case the presystolic rumbling murmur is due to a true mitral stenosis.

CASE VII.—L. R. Disp. No. 22,264 E; white; age, 16.

Diagnosis.—Aortic insufficiency, mitral stenosis and insufficiency.

This child gives a history of acute rheumatic fever seven years ago, followed by chorea. Since then she has had some dyspnœa when lying flat on her back and on exertion.

She is fairly well developed, but shows slight clubbing of the fingers.

Examination of Heart.—The point of maximum impulse is in the V interspace, 8.5 cm. from the mid-sternal line.

The impulse at the apex is forcible and heaving, and is preceded by a short, well-marked thrill. There is no diastolic thrill at the base.

The cardiac dulness extends 11 cm. to the left in the V interspace, 3.5 cm. to the right in the IV interspace, and above to the upper margin of the third rib.

On auscultation at the apex there is heard a very sharp, loud, pounding first sound, preceded by a short, rumbling, crescendo murmur. There is also heard a soft, blowing systolic murmur. At the apex no mid-diastolic murmur is audible, but as one passes inward a blowing prolonged diastolic murmur becomes audible, quite distinct and different from the rumbling presystolic murmur heard outside this point. The blowing diastolic murmur increases in intensity as one passes inward, and reaches its maximum at the left sternal margin in the III interspace. It is heard faintly as high up as the sterno-clavicular articulation, and as far to the right as the parasternal line at the third rib. In the aortic area there is also heard a soft blowing systolic murmur, transmitted upward to the sterno-clavicular articulation, but not well heard in the vessels of the neck. Passing from the sternum toward the left, the blowing diastolic murmur becomes fainter and fainter, and is almost inaudible in the mammillary line. It again increases in intensity, however, and about 5 cm. outside the mammillary line the murmur again becomes quite loud. From this area of greater intensity the murmur diminishes as one passes in all directions, but it is audible as high up as the apex of the axilla, and outward to the mid-axillary line.

This case also shows the wide distribution of the aortic diastolic murmur and the area of increased intensity in the axilla as shown in some of the previous cases.

CASE VIII.—H. S. White; occupation, laundryman.

Diagnosis.—Aortic insufficiency, mitral stenosis and insufficiency.

Examination of Heart.—The point of maximum impulse is in the IV interspace in the mammillary line, 11 cm. from mid-sternal line. The impulse is forcible, punctate.

On palpation over the apex the impulse is sharp and thudding, and there is a slight pre-systolic thrill.

The cardiac dulness extends 12.5 cm. to the left at the fifth rib, and 5 cm. to the right at the fourth rib.

On auscultation at the apex the first sound is sharp and snapping, and there is a loud pre-systolic rumbling murmur running up to and ending in the sharp first sound, having a crescendo character. Passing from the point of maximum impulse in all directions a distance only as great as the diameter of the bell of the stethoscope, the rumbling pre-systolic murmur is lost, and there becomes audible a blowing diastolic murmur heard throughout the whole of diastole. This blowing diastolic murmur is heard over the precordium, in the lower left axilla, and along the right margin of the sternum as high up as the I interspace, as shown by the dotted line in the photograph. This murmur, however, is everywhere very soft and faint except in the two areas about to be mentioned. At the sternal articulation of the fourth left rib, and over the IV interspace at the sternal margin the murmur is quite loud and blowing. Over the sternum and in the oartic area it is very faint. Passing to the left from the point of maximum impulse the murmur rapidly becomes much fainter and is barely audible over the body of the heart. Passing outward, however, it increases in intensity until a point is reached 3.5 cm. outside the point of maximum intensity, where the murmur is heard with greatest intensity, much louder than at the sternal margin. The murmur here has exactly the same quality and the same time as that at the sternal margin, and is directly continuous with it. It seems almost certain that this murmur is produced at the aortic valve, and is a true aortic diastolic murmur, even though it is heard with greater intensity in the axilla than it is at the sternal margin. The character of the pulse, which is markedly collapsing, also renders it quite certain that this patient is suffering from a true aortic insufficiency.

This case shows very well the axillary distribution of the aortic murmur, and shows that in certain cases the murmur may be more intense in the axilla than it is in the region of the sternum.

CASE IX.—H. B.

Diagnosis.—Aortic insufficiency and mitral insufficiency. Mitral stenosis (?).

Examination of the Heart.—The cardiac impulse is diffuse in the V. interspace, just outside the mammillary line.

The cardiac dulness extends to the nipple on the left, to the sternal margin on the right, and above to the second rib.

At the apex the impulse is sharp and forcible, and is preceded by a well-marked thrill. There is no diastolic thrill at the base.

On auscultation at the apex the first sound is loud and booming, is accompanied by a blowing murmur, which is transmitted into the axilla, and is preceded by a loud, rumbling crescendo

murmur, ending in a loud, sharp first sound. This rumbling presystolic murmur is transmitted outward as far as the anterior axillary line. No blowing diastolic murmur is heard in the axilla, but as one passes inward such a murmur becomes audible when the parasternal line is reached. This murmur is well heard over the sternum and in the aortic area, but is of greatest intensity at the left sternal margin at the level of the third rib. It is not transmitted widely to the right. In the aortic area there is also heard a soft blowing systolic murmur.

In this case there is no axillary distribution of the aortic diastolic murmur, the murmur being transmitted to the left only as far as the parasternal line.

Case X.—D. G. Gen. No. 60,219. Med. No. 21,423. White; age, 18; occupation, tailor.

Diagnosis.—Aortic insufficiency.

This patient gives a history of an attack of acute articular rheumatism eight years ago. Shortly after this he was examined at the Johns Hopkins Hospital Dispensary, and was found to have a blowing diastolic murmur at the base of his heart, best heard in the III left interspace. Since then he has had no symptoms referable to his heart. During the present admission he has suffered from scarlet fever from which he is now convalescent. He has also had an acute nephritis, acute arthritis, acute tonsillitis, and adenitis. From all these complications he is now convalescent.

At present he is suffering from no symptoms referable to his heart. The vessel wall of the radial is not palpable.

Examination of Heart.—The point of maximum impulse is in the V interspace, 9 cm. from the mid-sternal line. The dulness extends to this point on the left, and reaches 4.5 cm. to the right.

On auscultation the sounds at the apex are clear and sharp. Over the sternum, and extending to the left about 2 cm. outside the parasternal line, there is heard a blowing diastolic murmur with greatest intensity over the sternal articulation of the third left rib, and in the III left interspace at the sternal margin. It is transmitted upward only to the level of the second rib. No other murmurs are audible. Change of posture does not alter the distribution of the murmur except that when the patient is upright, it is heard a little lower, and a little farther to the left, than when he is in the recumbent position.

This patient showed a well-marked aortic diastolic murmur, which, however, was not transmitted to the apex or outside of it. He exhibited all the other features of aortic insufficiency, and had previously been treated in the hospital for broken compensation. At the time of examination, however, he was at work, and there were no signs of disturbed compensation except slight shortness of breath on exertion. This case does not show the axillary distribution of the aortic murmur, and there is no Flint murmur.

Case XI.—L. B. Gen. No. 61,257. Med. No. 21,742. Disp. No. 8791 E. Black; age, 51; occupation, tailor.

Diagnosis.—Aortic insufficiency.

This patient complains of "rheumatism and cough."

For the past four years he has had pain in the joints, but no acute attacks of acute rheumatic fever. He had gonorrhœa 20 years ago; he has taken alcohol in moderation.

He now complains of the pains in the joints, which have been present for four years. During the past three weeks he has had a cough.

Examination of Heart.—On auscultation at the apex the first sound is entirely replaced by a very soft blowing murmur, the second sound is much louder and accentuated, and on cursory examination it might very well be mistaken for the first sound, and the blowing murmur be considered as diastolic. The blowing systolic murmur is heard loudest in the IV interspace in the parasternal line, and is transmitted only a short distance outside the point of maximum impulse. At the base there is heard a prolonged blowing diastolic murmur, loud at the aortic area, most intense along the left margin of the sternum. It is heard faintly throughout the right side of the chest. The murmur is well transmitted outward along the left margin of the heart, and is heard throughout the entire left axilla as far as posterior axillary line. At the apex this diastolic murmur is not audible, and in the region of the nipple it is quite faint, but is louder in the axilla. Passing outward into the axilla in the II, III and IV interspaces, however, the murmur does not become fainter and then louder, but is heard with a gradually diminishing intensity.

In this case of pure aortic insufficiency the murmur is well heard in the axilla, but it seems to be a direct transmission along the left margin of the heart. There is no Flint murmur.

Case XII.—D. H. Gen. No. 62,274. Med. No. 22,098. Black; age, 37; occupation, laborer.

Diagnosis.—Aortic and mitral insufficiency.

This patient gives a history of acute rheumatic fever five years ago. He had syphilis five years ago; has been a hard worker, and a steady drinker. One year ago he began to have shortness of breath.

On examination there is very slight thickening of the vessel walls. While this is definite, it is thought that the cardiac lesion is probably of rheumatic origin.

Examination of Heart.—The point of maximum impulse is in the VI interspace, 13.5 cm. from mid-sternal line. There is ascites at present and the heart is pushed upward and to the left. The area of cardiac dulness is difficult to outline on account of the thick œdematous chest wall. The dulness extends, however, at least 6 cm. to the right of the mid-sternal line. There is no thrill. On auscultation at the apex both sounds are audible and with the first there is a loud rough systolic murmur, slightly musical in character, transmitted into the axilla. At the apex during the entire diastole there is a loud, rough, prolonged, slightly musical murmur. Passing inward the murmur is faint in the region of the nipple, but it again becomes louder, more intense, and of a musical character. It has very great intensity along the left margin of the sternum, where it has a distinct sawing character. Passing outward the murmur becomes fainter until the anterior axillary fold is reached. Here it again becomes loud and musical. In a small area, 6-7 cm. outside nipple, in anterior axillary line, over a small area hardly larger than the bell of the stethoscope, the diastolic murmur is of very great intensity, prolonged and musical in character, and the intensity diminishes as one passes from this point in all directions. The intensity of the murmur here is not quite so great as it is along the sternal margin, but the difference is not very great. At the sternal margin the murmur has more of a sawing character than it has in the axilla. It is heard faintly throughout the left back. There is no Flint murmur.

This case shows very well the axillary distribution of the murmur, and also the area of secondary intensity in the axilla.

Case XIII.—P. C. Disp. No. 19,992 E. White; age, 36 occupation, tailor.

Diagnosis.—Aortic insufficiency.

This patient gives no history of acute illness except measles when a child. There has been no acute rheumatic fever. The alcoholic history is negative; he denies all venereal infection.

He has had no cough, no dyspnœa on exertion. He has had some slight gastric disturbance, for which he comes to the dispensary seeking relief. The cardiac disturbance was discovered during the routine examination.

The patient is a well-nourished man; radial vessels not sclerosed, and the physical examination is negative except for the changes found in the heart.

Examination of Heart.—The point of maximum impulse cannot be seen or felt.

The area of cardiac dulness reaches 12 cm. to the left in the V interspace, and 4 cm. to the right in the IV interspace.

At the apex the sounds are best heard over the fifth rib just inside the mammillary line. The first sound is a little rough, accompanied by a short and faint systolic blowing murmur. The second sound is snapping. Passing inward and upward, a blowing diastolic murmur becomes audible, which has its maximum intensity in the IV left interspace at the sternal margin. Even here the murmur is soft and not of great intensity. Passing outward above the nipple the murmur becomes very faint, but is even here audible in the area indicated by dotted lines in the photograph, but it again becomes more intense in the axilla, and is heard over a wider area. In an area over the pectoral fold, 7-8 cm. from the nipple, the murmur is heard with greatest intensity, louder than it is at the sternal margin.

This case shows in a very striking way the axillary diastolic murmur which we have been describing. Here is seen the dumb-bell shape area of distribution, which is, however, much less typical than in the other cases. There are two areas of greater intensity, one at the sternal margin, and the other in the axilla, with the murmur decreasing in intensity as one passes from each of these points in all directions. No Flint murmur is present.

CASE XIV.—H. S. Gen. No. 63,562. Med. No. 22,524. White; age, 58; occupation, saloon-keeper.

Diagnosis.—Aortic and mitral insufficiency.

This patient gives a history of repeated attacks of gonorrhœa, the first one 38 years ago. There is a definite history of syphilis. He has taken alcohol in excess up to the past few years. There is no history of acute rheumatic fever.

At present he complains of shortness of breath and palpitation of his heart, and at times precordial pain, especially on exertion.

He is a moderately well-nourished man; no œdema of feet. There is marked thickening of the arterial walls. The pulse is collapsing.

Examination of Heart.—The point of maximum impulse is in the V interspace, 11.5 cm. from mid-sternal line. There is some precordial bulging, maximum over the third rib.

The transverse area of cardiac dulness measures 18 cm.

On auscultation at the apex the first sound is rather muffled and prolonged and accompanied by a soft systolic murmur. In the area about the apex, outlined by the dotted line, there is heard a soft, but distinct, prolonged, blowing diastolic murmur. It is not rumbling in character, except just at the point of maximum impulse, over an area not larger than the bell of the stethoscope, where it is a little rumbling and more crescendo in character. The murmur in the axilla is loudest in the VII interspace, 7 cm. outside the point of maximum impulse, as shown in the diagram. It is not widely transmitted throughout the axilla. Passing inward the diastolic murmur becomes inaudible, but is again heard over the wider area included by the larger dotted line on the photograph. The murmur over this entire region is quite soft and faint, except in the area shown by cross line in the I and II interspaces to the right of the sternal margin, where it is quite intense and loud. In this area there is also heard a loud systolic murmur, which, however, is not well transmitted into the vessels of the neck, and the character of the pulse does not indicate an oartic stenosis.

In this case the aortic diastolic murmur is everywhere quite soft, but nevertheless it is heard outside the apex, and here there is a small area where it is heard with secondary intensity. This murmur about the apex is not a direct transmission outward, that is, it cannot be traced directly into the basal murmur, but nevertheless it has exactly the same character, and is of the same time, and we feel that it must be due to the regurgitation of blood through the aortic orifice.

CASE XV.—R. R. Gen. No. 62,850. Med. No. 22,287. Black; age, 60; occupation, cook.

Diagnosis.—Aortic insufficiency; gout.

This patient gives a marked alcoholic history, and probable history of lues. He has had attacks of gout during the past year. There is no history of acute rheumatic fever.

He is a well-nourished man, at present having some dyspnœa. The radial vessels are moderately sclerosed.

Examination of Heart.—The point of maximum impulse is in the VI interspace, 13 cm. from the mid-sternal line.

The dulness reaches 3.5 cm. to the right over the fourth rib. There is also slight impairment of the percussion note over the manubrium, and in the I and II right interspaces.

On auscultation at the apex the first sound is well heard, and is accompanied by a soft systolic murmur. The second sound is well heard. There is barely audible here a very soft diastolic murmur, which is transmitted only over a limited area about the apex, as shown by the dotted line. This murmur is heard loudest just above and to the outer side of the point of maximum impulse. It is faint, but well heard, and is soft, prolonged, blowing, and has exactly the same character and time as has the diastolic murmur heard in the oartic area. Inside the point of maximum impulse and over the body of the heart this murmur is entirely inaudible until one approaches the border of the sternum. It becomes loudest in the II right interspace at the margin of the sternum, though even here it is quite faint and distant.

In this case the aortic diastolic murmur is faint but very distinct, and the other features of the case render it quite certain that the patient is suffering from insufficiency of the aortic valve. Notwithstanding the faintness of the murmur it is heard outside the apex, and there is a small area of secondary intensity as shown by the diagram. It might be objected that the murmur heard about the apex is a Flint murmur, but the murmur has none of the characteristics of that heard in mitral stenosis, and it is exactly like the murmur of aortic insufficiency heard at the base.

CASE XVI.—W. B. Disp. No. 23,423 E. White; age, 47; occupation, can-maker.

Diagnosis.—Aortic insufficiency.

This patient has been a hard worker, and has a marked alcoholic history; has usually taken beer. He gives a history of having had several attacks of gonorrhœa and syphilis 22 years ago.

The present illness began with cough and pain in the chest two months before admission to the hospital.

There is not a marked grade of cardiac incompetency. There is slight shortness of breath.

His vessels show a marked grade of sclerosis. The pulse is 80, regular, and there is considerable throbbing of the vessels, a well-marked Corrigan pulse.

Examination of Heart.—The point of maximum impulse is in the V interspace, 14 cm. to the left of the median line. The cardiac dulness extends 1.5 cm. outside this point, and reaches 3 cm. to the right in the IV interspace.

On auscultation at the apex the first sound is loud, and is accompanied by a slight, short, squeaking murmur. Preceding the first sound there is a short, rumbling murmur, crescendo in character, and ending in the first sound. Passing inward, this rumbling character disappears, and there is heard a blowing diastolic murmur extending through the entire diastole. This murmur is heard over the entire cardiac area, and is transmitted throughout the left side of the chest and axilla, as shown in the diagram. While it is heard continuously in passing from the sternum to the axilla, nevertheless, in a zone 3 to 4 cm. wide in the region of the nipple and about the point of maximum impulse it is heard extremely faintly—is barely audible. Outside this area in the axilla it again becomes louder, and is heard quite loudly, almost with maximum intensity, in the area shown by cross lines on the photograph. It is also heard loudly, slightly louder than in the above-mentioned area, in the aortic area, in the III interspace close to the sternum. The slight presystolic rumbling murmur (Flint) is heard only over a small area at the point of maximum impulse, and has an entirely different quality from the loud, prolonged, blowing diastolic murmur.

This case shows very well the axillary diastolic murmur with an area of secondary intensity in the axilla.

Case XVII.—J. K. Med. No. 21,386. Gen. No. 60,112. White; age, 22; occupation, painter.

Diagnosis.—Typhoid fever, psychosis, aortic insufficiency.

The patient gave no history of rheumatic fever or chorea. The venereal history is negative, as is also the alcoholic history. The patient was admitted early in an attack of typhoid fever, and the aortic insufficiency was discovered at the examination. The patient passed successfully through the fever, the only complication being a moderate psychosis. The following examination of the heart was made during convalescence.

Examination of Heart.—The point of maximum impulse is in the V interspace, 9 cm. from the mid-sternal line. The dulness does not extend outside this point, and reaches only to the right margin of the sternum.

On auscultation at the apex both sounds are well heard, loud and sharp. There is slight reduplication of the first sound. In the small area just about the point of maximum impulse, shown in the photograph by the continuous line, there is heard a well-marked, presystolic, rumbling murmur (Flint). There is also audible a very soft systolic murmur, which is transmitted outward to the anterior axillary line. At the base both sounds are well heard; there is slight accentuation of the pulmonic second sound. Over the areas indicated by the dotted lines there is heard a loud blowing diastolic murmur, extending throughout diastole, of maximum intensity over the area shown by cross lines in the diagram. This murmur is audible outside the apex, as indicated, but it is soft, and there is no spot of increased intensity. Here it has exactly the same character as that at the base and over the body of the heart, and has not at all the rumbling character of the pre-systolic murmur just about the point of maximum impulse.

This case shows very well the axillary transmission of the murmur, but there is no point of maximum or secondary intensity, as seen in some of the other cases. There is a well-marked Flint murmur.

These cases, we think, are sufficient to show the frequency with which the aortic diastolic murmur is transmitted to the axilla. They also show that at times the murmur may be heard over a small area in the axilla with secondary, or even maximum intensity. We have not attempted to make a statistical study, but desire only to call attention to the points which have been neglected or overlooked by writers on this subject.

The above cases are of both the arterio-sclerotic and endocarditic types. At least five of them are certainly not of infectious origin, but the cardiac lesion is simply a part of the general arterio-sclerotic process. There seems to be no difference in the distribution of the aortic murmur in the two types of cases, the murmur being heard in the axilla equally well in both.

In all but two of the seventeen cases the diastolic murmur has been heard outside the apex. Further studies may show that this slightly exceeds the real frequency, but we are convinced that the murmur is almost always audible in the axilla.

In eleven of the cases the murmur has been heard with increased intensity over an area outside the apex. In other words, passing to the left from the area of greatest intensity in the region of the sternum, the murmur diminishes in intensity and then, outside the apex or in the axilla, again becomes louder. In two cases (IX and XIII) the murmur was louder over this axillary area than it was at the sternal margin or in the aortic area. In Case III, also, the axillary murmur was almost, if not quite as loud, as it was at the base. In two cases (XIV and XV) there were two distinct areas over which the murmur was audible, and between which the murmur could not be traced.

The area outside the apex over which the murmur is heard with increased intensity is well shown in the photographs. It will be seen that in most instances this area is 3 to 4 cm. outside the point of maximum intensity, and usually in the interspace above it, though sometimes this area is over the same interspace, or even at a slightly lower level.

Corresponding to the findings of most other observers, the point of maximum intensity is most frequently at the left sternal margin (in nine of our cases). Five times it was at the right of the sternum, twice over the mid-sternum, and once, as mentioned, in the axilla.

In eleven of the cases there was a rumbling presystolic murmur heard at the apex, though in some it was very faint. In two a definite diagnosis of mitral stenosis could be made, and in five others the physical signs, history and general features rendered it probable that a true mitral stenosis was present, though in most of them the possibility that the murmur was only a Flint murmur could not be excluded. In four the presystolic rumbling murmur seemed to be quite definitely that described by Flint. In six, however, there was no suggestion of a rumbling presystolic murmur at the apex, but, nevertheless, the blowing aortic diastolic murmur was heard at and outside this point in all but one.

We foresee that the chief objection that will be raised to our description of the axillary aortic diastolic murmur will be that we have been listening to the diastolic mitral murmur (either a true stenotic murmur or a Flint murmur)

CASE 1.

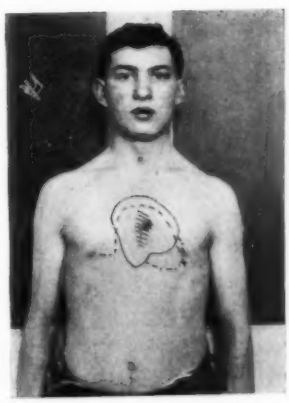
CASE 3.

CASE 4.

CASE 5.

CASE 6.

CASE 8.



CASE 10.

CASE 13.

CASE 14.

CASE 15.

CASE 16.

CASE 17.

which is transmitted into the axilla, or where we have described a diastolic murmur about the apex not directly continuous with the murmur at the base, that we have been listening to a Flint murmur which has been more than ordinarily blowing in character. We feel convinced, however, that such objections are not valid. The fact that the murmur described has been of exactly the same kind and quality as that heard at the base makes it seem almost certain that both have an identical origin. Moreover, these findings can be easily confirmed by any one who will carefully study and map out the distribution of the aortic diastolic murmur.

It is probable that the reason why so little attention has been paid to this murmur is that in many cases the diastolic murmur heard in the axilla has either been considered to be a Flint murmur, or even, where it has not been carefully observed, it has been considered to be systolic in time, as in the cases previously mentioned. We have no theories to offer as to why it should be louder at and outside the apex in certain cases than it is along the left sternal margin, though it is quite easy to understand why it should be fainter over the body of the heart. Whether, as Foster maintained, the aortic murmur is more likely to be transmitted to the apex in cases where the posterior aortic segment is involved than in cases where no such involvement is present, is very doubtful. We have had an opportunity to examine the hearts after death in two instances in which the axillary distribution and axillary intensification were present during life: In each of these the anterior cusps were involved, but the posterior cusp were quite normal.

In the present paper we have attempted to show that it is the rule rather than the exception that the diastolic murmur of aortic insufficiency is transmitted loudly to the axilla, and furthermore that it is not a transmission of the murmur towards the apex in the general acceptance of this statement, but that in most cases there is a second area of greater intensity above and outside the apex, and that while in many cases the murmur is heard continuously as one passes outward from the sternum to the axilla, becoming fainter and fainter, in other cases the murmur becomes inaudible in the region of the nipple, and is again heard in the axilla, or in rare cases the murmur may be entirely inaudible over the heart and at the base, and only be heard, at times at least, outside the apex. For this axillary diastolic murmur we would suggest the term *aortic axillary diastolic murmur.*

CERTAIN CLINICAL FEATURES OF CARDIAC DISEASE.[1]

By G. A. GIBSON, M. D., LL. D.
Physician to the Royal Infirmary, Edinburgh, Scotland.

Mr. President, Ladies and Gentlemen: Allow me in the first place to express the pleasure which has been afforded me in having the opportunity, through the kind invitation of my friend, Professor Barker, of meeting you on this occasion. To be requested to make any remarks upon cardiac disease before this society, the members of which have done so much admirable work upon many aspects of the circulation, in health as well as in disease, is indeed a distinction only mitigated by the feeling that it will be difficult to advance anything that is not already well known to you. The title which has been suggested for my address allows, however, a wide latitude of which advantage will be fully taken. The result will undoubtedly be that my remarks will be somewhat desultory, but they will be essentially clinical.

Let me direct your attention in the first place to some of the nervous features associated with that complex of symptoms which receives the name of angina pectoris. It is quite unnecessary to dwell upon the main circulatory disturbances, but it may be well to mention in passing that the arterial pressure may be high or low, and the heart and vessels may show very great, or extremely small, departures from normal conditions. The pain, which is the central fact in the nervous symptoms, is essentially of the variety which has been termed "referred" or "somatic." For its thorough comprehension we are mostly indebted to the observation of Mackenzie and Head, who have not merely traced out its distribution in a way previously unapproached, but have furnished an explanation as beautiful as it is complete. The main situation of the pain is in the region of the præcordia, whence it radiates towards the shoulder and down the arm, as well as, less frequently, up the side of the neck and head. In disease affecting the left side of the heart primarily, the pain is experienced mostly, or solely, on the left side; but, as has been shown by Morison and myself, when the structural changes are confined to the right side of the heart the painful symptoms are found in the corresponding areas. The changes in sensibility are found most typically along the course of the distribution of the ulnar nerve and its connections; that is to say, they correspond to the brachial roots connected with the seventh and eighth cervical segments, and the first thoracic segment, of the cord. One of the modern additions to our knowledge of these nervous symptoms was made by Mackenzie, when he directed attention to the fact that there is hyperæsthesia over the areas in which subjective pain is experienced. Careful analysis of the phenomena has since his observations were published, fully confirmed their accuracy. It has for some time been recognized that the numbness, which has for long been described by sufferers from the affection, is oftentimes attended by diminution of sensibility, which may reach such a degree as to attain complete anæsthesia.

The explanation of these phenomena, which has been given

[1] An address delivered before The Johns Hopkins Hospital Medical Society, October 1, 1908.

by Head is most interesting. The sensory impulses arising in connection with the heart or aorta pass to the central nervous system by means of the afferent cardiac nerves to the ganglia of the sympathetic system, particularly the middle and inferior ganglia, as well as the stellate ganglion, whence they are transmitted by the rami communicantes to the spinal cord. The impulses pass thence up the cord to the receptive areas of the brain, but as these, by the course of education, have been taught to associate disturbances in the spinal region which is affected with those areas of the surface of the body with which the sensory spinal nerves are in relation, the source of the disturbance comes to be associated with the nerves going to these superficial areas. Hence it is that the pain in angina pectoris centers so largely in the ulnar distribution. The explanation of the anæsthesia is, undoubtedly, that long-continued over-use of any nervous structure is apt to lead to exhaustion and paralysis.

It has been shown by Eichhorst and myself that wasting of the muscles, and changes in the texture of the skin are to be made out at times in the regions associated with the painful symptoms. It has, further, been demonstrated by myself that when muscular atrophy is found the myotatic irritability may be increased, exactly as is the case in arthritic muscular atrophy, while the electric reactions show a distinct increase, both as regards faradism and galvanism.

The eyes occasionally show alterations, to which it is my wish to direct your attention. The eye on the side to which the pain is mainly, or entirely, confined, may be more prominent than its fellow; while the aperture between the eyelids may be increased in width, and the pupil may at the same time be dilated. The explanation of these symptoms is very simple. If there be sufficient stimulation of the sympathetic system to reach the first cervical ganglion, the impulses may pass from it to the nervous filaments which control the muscle of Müller at the back of the orbit, to those which innervate the unstriped fibers of the levator palpebræ superioris, and to those which cause contraction of the radiating fibers of the iris. In this way protrusion of the eyeball, elevation of the upper lid, and dilatation of the pupil result.

These varying changes may be found in varying degrees, more or less of them being associated in any given case; at times many, or even all of them, may be present in the same individual, and in order to illustrate these remarks, let me refer very briefly to a most interesting patient who was under my care a few years ago, when Dr. H. A. Stewart, who is at present attached to this medical school, was my house physician. The patient was a man of middle age, engaged in the hard work of an unskilled laborer, who came into my ward complaining of agonizing pain in the breast and left arm. He was found to have no evil hereditary tendencies, while his past history showed no specific disease of any kind. His habits as to alcohol and tobacco did not seem to merit reprobation. He had been attacked quite suddenly by the symptoms when on his way to work one morning, and has been compelled to desist from making any further attempts at exertion. The patient presented a healthy appearance with a fresh complexion, but an anxious expression. Inspection of the face showed dilatation of the left pupil, prominence of the left eye, and increased width of the aperture between the lids. The conjunctival reflex was lively in both eyes and the iris rsponded promptly on both sides, both to light and to distance, the left pupil always remaining larger than the right. The pain was distributed over a wide area, extending over a great part of the left half of the chest, and up to the lower part of the neck from the middle line in front to that behind. As regards the arm, there was no pain over the distribution of the ulnar nerve, but it involved all the rest of the left arm down to the wrist. Over the whole of this area there was a high degree of hyperæsthesia; at the wrist there was a region about an inch broad encircling the limb with the exception of the ulnar area; this area was insensitive to painful impressions, but the patient could feel perfectly well when he was touched. Below this, with the exception of the region supplied by the ulnar nerve, every form of sensibility was completely abolished. In this case, therefore, on the outer aspect there was subjective pain and objective tenderness as far as the wrist, while at and below it, there was, firstly, analgesia without anæsthesia, and still lower, both analgesia and anæsthesia.

The left arm and forearm were somewhat smaller than those on the right side, and their power was notably diminished. The grip of the left hand was less than half that of the right. The muscle and tendon responses were, on the other hand, considerably greater in the left than in the right arm; and both to faradism and to galvanism, the muscles responded with a very much smaller current than in the case of the right arm. We could not, in this case, be certain that there was any alteration in the nutrition of the skin.

There was a certain degree of thickening of the superficial arteries, and the arterial pressure was high; unless my memory plays me false, it was, on admission, about 200 millimeters of mercury. During the paroxysms of the pain it appeared to be even higher, but for obvious reasons it was not specially measured at such times. When Dr. Stewart was engaged one day taking some tracings with the sphygmograph, an attack appeared, and the curve, which had shown the ordinary features of a somewhat high-pressure pulse, at once became much smaller in amplitude with its tidal wave higher up on the descending limb; in other words, it was a tracing of a pulse of higher pressure than before. The administration of nitrite of amyl promptly relieved the patient (as it always did during an attack), and brought about a return of the pulse to lower pressure. The heart was slightly enlarged, and the aortic second sound was considerably increased in intensity, but there were no other objective evidences of cardiac disease.

The ocular phenomena can be clearly seen in the pictures thrown on the screen, while the sensory disturbances which were traced upon the surface of the patient (who was afterwards photographed), have also been shown along with photographs of the pulse tracings.

It is satisfactory to be able to state that by means of the nitrites and iodides the patient made an excellent recovery. For some slight ailment he presented himself in my ward during the present summer, and we were able to assure ourselves of this fact.

Disturbances of the rate and rhythm of the heart have during the last few years furnished a fertile field for discussion. It is obviously too large to enter upon fully on the present occasion, but there are some aspects of the subject to which you will, perhaps, allow me to direct your attention. Before entering upon any discussion of the clinical bearings of such disturbances it is necessary to turn briefly to the mechanism involved. The researches of Gaskell upon the connections of the auricle and ventricle, in which he was followed by a number of observers, showed the existence of the now well-known auriculo-ventricular bundle. The entire course and distribution of this interesting connecting link were unfolded by the indefatigable researches of Tawara, demonstrating that after passing down from the inter-auricular to the inter-ventricular septum, the branches of the bundle radiate outwards in every direction, and are continued as the Purkinje fibers which reach not merely the central portion of the long scroll of muscular tissue described by J. B. MacCallum, but also the musculi papillares. The later investigations of Keith have not merely amplified certain details of this arrangement of fibers, but have furnished an altogether new and most interesting addition to our knowledge. Keith has recently shown that at the junction of the sinus and auricle of the right heart there is a peculiar structure, consisting of undifferentiated muscular tissue containing a large number of nerve ganglia and nerve fibers.

Gaskell not only analyzed the functions of the heart wall, pointing out that it possessed rhythmicity, conductivity, excitability, contractility, and tonicity, but he showed that there was a natural block at the auriculo-ventricular junction—often since termed "Gaskell's bridge"—and he further brought the fact into prominence that this block could be increased by artificial means. His discovered that by section of the auriculo-ventricular bundle impulses could be partially or totally interrupted in their passage downwards. Erlanger, while attached to the Johns Hopkins University, introduced a most ingenious method of exerting pressure upon the bundle and discovered that every degree of block might be produced.

Bradycardia or abnormal infrequency of pulsation may be false or true; the former brought about simply by failure of some of the ventricular impulses to reach the periphery, so that the rate of the arterial pulse is less than that of the heart; the latter produced by infrequency of the heart itself. False bradycardia may be regarded simply as an expression of cardiac failure, and it is not my intention to deal with it on this occasion. True bradycardia may be total or partial; to the first group belong cases in which the entire heart, auricular as well as ventricular, participates in the reduced rate. Instances of pressure upon the vago-accessory fibers, of toxic influences affecting the nervous mechanism or the muscular structure, and degenerative changes affecting the heart wall are familiar instances in point. From the examination of the veins of the neck, of the peripheral arteries, and of the cardiac apex by the graphic method with simultaneous tracings, as well as by means of the fluorescent screen, we are able to determine that the auricles as well as the ventricles are involved in such cases. In the second group are instances in which only part of the heart is involved. Many observers have published cases illustrative of such conditions, and in their investigation we have been greatly assisted by the researches of Erlanger and Hirschfelder of your own school. The best known form of this type of disturbance is found in block at the auriculo-ventricular band in which tracings demonstrate the existence of auricular impulses which are not transmitted to the ventricles. This is also borne out by the results of auscultation, for the sounds produced by the auricles may be heard in the intervals between ventricular contraction. The use of the fluorescent screen further shows in such cases that the auricles contract without any following pulsation of the ventricles. It is a matter of much interest to see in such examples that the use of drugs, such as atropine, can influence the rate of the auricles without producing any effect on the ventricles. This ventricular bradycardia may be partial or complete. By the first of these terms a condition is defined in which some of the auricular impulses (usually every third one) pass to the ventricles and initiate corresponding contractions. The second phrase denotes the condition in which there is total dissociation of the movements of the auricles and ventricles, the two different levels of the heart each pursuing an independent rhythm of their own. The tracings which have been thrown on the screen amply demonstrate these different types. Let me add lastly in regard to this part of the subject that some of these tracings may be taken as proving that it is not always conductivity which is impaired, but that excitability may be lessened.

In this particular type of heart block, the localization of the lesion has been found in a considerable number of cases to lie in the auriculo-ventricular band. The first pathological evidence of this fact was furnished by Stengel of Philadelphia, but several others, almost immediately afterwards, were fortunate enough to meet with instances also. It is probable that ere long our information will be extended, and that we shall discover new facts which will more or less modify our views. A heart which was shown to me the other day by Professor Adami and Dr. Klotz, of Montreal, concerning which the entire facts will be published by Professor Martin, in whose care the patient was, will certainly, to some extent, reopen the questions involved.

It is probable that a block may take place higher up—at the sino-auricular junction. Hering, Wenckebach, and Hirschfelder have already expressed their belief in such a possibility, and one of the tracings which has just been shown, and which was previously published by Ritchie and myself, seems most easily explicable on the supposition that certain of the smaller waves which occur amongst, but do not accord with, the rhythm of the auricular impulses, are produced by

the sinus contraction. It is an interesting fact that the movements of the auricles in such cases are remarkably regular; when we find other waves appearing irregularly amongst them we must seek for some other cause, and this is most likely to be found in a pulsation of the sinus which has not been followed in the normal manner by the auricular systole.

An abnormal increase in the rate of pulsation commonly known in modern times as tachycardia may apparently be produced by neurotic, toxic, or degenerative influences, but our comprehension of the subject is not yet so advanced as in the case of the conditions which have already been discussed. From the tracings which have been shown, you will readily accept the statement that the increased rate is largely dependent upon the presence of premature systoles. Whether the origin of this change may be in part due to diminished influence of the inhibitory, or to increased activity of the accelerator nerves is uncertain, but we may assume in all such cases an increased production of the rhythmic stimulus at the sino-auricular junction of Keith, which must be regarded as the pace-maker for the whole heart, except in those cases where the impulses are blocked. There is, however, nothing now more certain than the fact that the ventricle may initiate a rhythm of its own, and one of the tracings which has just been brought before you shows most distinctly contractions of the ventricles in the total absence of all auricular movement.

Will you allow me, Mr. President, to express to you, as representing on this occasion the university, my great satisfaction in being permitted to witness the wonderful strides which you have made. Coming amongst you for the first time, the noble words which Huxley uttered at the opening of this great institution two decades ago naturally occur to my memory:

"Truly America has a great future before her; great in toil, in care, and in responsibility; great in true glory if she be guided in wisdom and righteousness; great in shame if she fail. I cannot understand why other nations should envy you, or be blind to the fact that it is for the highest interest of mankind that you should succeed; but the one condition of success, your sole safeguard, is the moral worth and intellectual clearness of the individual citizen. Education cannot give these, but it may cherish them and bring them to the front in whatever station of society they are to be found; and the universities ought to be, and may be, the fortresses of the higher life of the nation.

May the university which commences its practical activity to-morrow abundantly fulfil its high purpose; may its renown as a seat of true learning, a center of free inquiry, a focus of intellectual light, increase year by year, until men wander hither from all parts of the earth, as of old they sought Bologna, or Paris, or Oxford."

How abundantly those responsible for the administration of the university have fulfilled their trust, and how loyally those who have worked under their auspices have performed their duties, can be read by all in the important series of observations which have enriched modern scientific literature; but this is not all, for the free spirit of inquiry which you have shown has been wafted to all lands, and has awakened everywhere a sympathetic response. The hopes which Huxley so generously expressed have been far more than realized, and seekers after truth from many countries have directed their footsteps hitherwards. Amongst these have been some of my own particular friends, for your kindness to whom you will perhaps allow me to tender my warm acknowledgments. That the university may continue to foster the higher education and to increase its usefulness is the fervent hope of all your friends, amongst the warmest of whom, you will allow me to enroll myself.

THE PRACTICAL VALUE OF THE DEMONSTRATION OF *SPIROCHÆTA PALLIDA* IN THE EARLY DIAGNOSIS OF SYPHILIS.

By J. T. Geraghty, M. D.

Instructor in Genitourinary Surgery, The Johns Hopkins University.

It is over three years since Schaudinn and Hoffman first reported the finding of *Spirochæta pallida* in smears made from the primary and secondary lesions of syphilis. The announcement was very modest and made no exaggerated claims, simply stating that the writers had found in syphilitic lesions a spirochæta which was morphologically distinct from other forms. They also described another form of spirochæta which was non-pathogenic and sometimes resembled quite closely the *S. pallida,* and to this organism they gave the name *Spirochæta refringens.* This latter organism is important on account of its frequent occurrence in superficial ulcerations and its close resemblance at times to the syphilitic organism.

Since the first publication of the findings of Schaudinn and Hoffman an enormous amount of work has been done and the observations of the different workers have almost unanimously confirmed the findings of the discoverers of the organism. Although the *S. pallida* has so far not been definitely grown on artificial media, and consequently all of Koch's requisites have not been fulfilled, nevertheless the evidence in favor of its being the etiological agent in syphilis has so accumulated that it is generally accepted as such by those most competent to judge. The evidence may be briefly summed up as follows:

(1) The presence of the *S. pallida* and the absence of any other organism in the still-born, dead of heriditary lues. (2) The number of *S. pallida* corresponds to the extent of the disease in the syphilitic organ and their absence or scarcity in organs not diseased has been commonly noted by those who have investigated the matter. (3) The almost constant finding of the *S. pallida* in the primary lesions of man and monkeys. Schaudinn found it 70 times in his last 70 cases of

primary and secondary syphilis. Sobernheim in 50 cases of primary and secondary syphilis found the *S. pallida* in every case. Metchnikoff and Roux in their experimental work on apes found the *S. pallida* 23 times in 31 infected animals and they have never found any spirochætæ other than the *S. pallida* in their animal innoculations. Hoffman found the *S. pallida* 14 times in 14 chancres. Mulzer found it 20 times in 22 syphilitic cases, and learned of 32 chancres in which it had been found 30 times. Flügel found it 28 times in 29 chancres. Herxheimer and Hübner 16 times in 17 cases of primary and secondary syphilis. I have quoted at considerable length these authors and their positive findings in order to show in what a small percentage of cases of syphilis negative findings occur in the experience of skilled observers. (4) The fact that the *S. pallida* has never been found except in syphilis. (5) The finding of the organism in syphilis in the most distant countries. (6) The lesions which are the most contagious in acquired syphilis, the chancre, condyloma and mucous patch are the ones in which the *S. pallida* are the most numerous and most often found. (7) They have been found in all the lesions of syphilis, in the blood in the secondary stage and even in the urine in a case of syphilitic nephritis, etc. (8) Mercury, which is the only drug that can cure syphilis, causes the very rapid disappearance of the *S. pallida* from the local lesions.

In order to determine if the *S. pallida* could be demonstrated with sufficient ease in the primary sores to be of any practical value and whether it was possible to definitely distinguish it from the commonly associated non-pathogenic *S. refringens*, a systematic examination of all venereal sores in patients coming to the genitourinary dispensary of The Johns Hopkins Hospital was begun over a year ago.

Those who see many venereal sores realize that in a great number of cases it is impossible to distinguish between chancroid and chancre. The time element is seldom of any value so that the period of incubation cannot be determined. The syphilitic sore may be nothing more than a tiny abrasion lasting only a few days and no suspicion of its true character entertained until it is subsequently revealed by the development of secondaries. Again and again we have seen apparently typical chancroids which afterward proved to have been syphilitic sores.

The diagnosis was so uncertain in many cases that it became a routine measure in our clinic to wait for the development of secondaries. It is unjustifiable upon mere suspicion or probability to condemn a patient to three or more years of vigorous mercurial treatment while on the other hand it seems only rational that the specific treatment should be begun as early as possible. Experience has shown that those cases in which an early diagnosis could be made from the primary sore and in which treatment was begun before the appearance of secondary lesions ran generally a mild course and according to Fournier were much less liable to tertiary accidents.

During the past year we have searched for *S. pallida* in nearly all of the venereal sores of patients coming to the genitourinary dispensary. We have examined about 150 cases in all; of this number 30 were syphilitic—they either had definite secondaries when first seen or subsequently developed them. Other syphilitic lesions such as condylomata, mucous patches and secondary skin eruptions were also searched for organisms. Comparatively few examinations of tertiary lesions were made as very little of this material presented itself in the clinic. Smears from the syphilitic organs of the still-born fœtus were examined in three cases.

The technique employed has been as follows: The sore is thoroughly cleaned with soap and water, and the surface of the sore rubbed with a piece of gauze until small bleeding points appear. After the surface of the lesion has been scraped in the manner above described the sore is firmly compressed between the thumb and forefinger. This firm pressure causes at first quite an ooze of blood, but if pressure is kept up an almost clear serum will soon exude. A clean slide is then rapidly swept across the exuding serum—usually from three to four smears are made from each sore. We have found the above method of making the smears very satisfactory and almost uniformly successful. The most essential step in the search for the *S. pallida* is the making of good smears and success or failure will depend largely upon it. The sores are thoroughly cleaned in order to eliminate as far as possible a contamination with the *S. refringens*, which is a surface grower, while the *S. pallida*, being a true parasite, is most apt to be obtained from the serum which is squeezed out of the living tissue. The same technique is employed in making smears from condylomata and mucous patches on the lips. Where the mucous patches are situated on the inner cheek, tonsils or fauces satisfactory smears are obtained with difficulty.

In obtaining smears from the skin lesions the superficial epithelium should be scraped off with a knife-blade until small bleeding points are visible. The serum is then squeezed from the abraided surface and the smears are made in the same manner as those from the chancre. When the skin lesion is a pustule the pus must be evacuated and the serum obtained as far as possible from the bleeding edge of the pustule. If an examination only is made of the pus or necrotic material it will be almost uniformly negative.

Similarly in searching for the organism in tertiary ulcers the importance of obtaining the material from the edge of the sore has been emphasized by Schaudinn himself.

Method of Staining.—In our earlier work the Giemsa stain was almost exclusively employed. The method of staining is as follows: The smears are first fixed in absolute alcohol for 20 minutes and are then ready for staining. We have found a dilution of one part of the stain to four of distilled water the most satisfactory. The slides are covered with this dilute stain (the dilution should be made fresh each time), a bell jar is placed over them to prevent evaporation and they are allowed to stain from 12 to 18 hours. It is difficult to overstain. The slides are then washed for a moment in running water, dried and are ready for examination. The *S. pallida* will be stained a pinkish tinge while the *S. refringens* takes a

deep blue. This differential staining, however, is not always absolute as occasionally blue staining *S. pallida* are encountered. Of late we have given up entirely the Giemsa stain and employed instead that of Hastings.

The method is as follows: The slides after being air-dried, are then covered completely with the stain, care being taken to keep the slides well covered with stain to prevent precipitation. After one minute distilled water is added until a metallic film forms and the smears are allowed to stain for five minutes more. They are then quickly washed in running water and dried. The *S. pallida* are usually stained a faint blue and occasionally have a pinkish tinge, while the *S. refringens* are stained a deep blue. The Hastings stain is to be preferred to the Giemsa as the method is simpler and the material can be stained in a few minutes. Furthermore, the staining is more intense with Hastings than with Giemsa. We have employed various silver staining methods, but have not found any of them as uniformly satisfactory as the Hastings. The principal objection to the silver method is the time which it consumes.

Our experience with the dark field illumination which enables one to see the organism in the fresh unstained serum is as yet too meager to permit comparison with the staining methods mentioned.

Differential Characteristics.—It may be well to enumerate some of the characters of the *S. pallida* which serve to distinguish it from the *S. refringens*. The *S. pallida* is extremely slender, of uniform caliber, except the ends which taper almost to an invisible point. The *S. refringens* is much thicker. The spirals vary in number from four to twenty or even more and the length of each spiral is about half the length of the spiral of the *S. refringens*. If the length of the spiral is two microns or over it is never a *S. pallida*. The spirals are fairly uniform, gradually and very slightly diminished in size from the center towards the end and are rather angular. The spirals of the *S. refringens* are more wool like. They are neither so uniform nor so numerous and are not so close set as are the spirals of the *S. pallida*. The *S. refringens* are readily stained by ordinary dyes.

Results.—A routine examination of all venereal sores of patients appearing in the clinic was undertaken. In all about 150 cases were examined and of this number 30 were syphilitic —they either had definite secondaries when first seen or subsequently developed them. In these 30 cases *S. pallida* were demonstrated in smears from the primary lesions in 27 cases. Every case in which the organism was demonstrated had definite syphillis when first seen or subsequently developed manifest symptoms. Specific treatment was not begun in many cases in which the organisms were found until definite evidence of the disease appeared, as we wished to test the reliability of our findings. In two of the three cases in which we failed to find the organism the sore had begun to heal. It has been our experience in other cases that where the primary lesion has begun to show signs of repair, it is frequently difficult to demonstrate the *S. pallida*. This of course is natural as the healing denotes the disappearance of the organisms from the lesion. In the third case, although numerous attempts were made to find the *S. pallida*, the results were always negative.

It is of some interest to note that in two preputial chancres in which the organism had been readily demonstrated it was impossible to find any organism in smears from the chancres 24 hours after they had been excised. The Giemsa stain was employed in staining the smears from the excised chancres. We have had a similar experience in smears made from the organs of three syphilitic fœtuses. Although a large number of slides were examined, in each case both the Giemsa and Hastings stain being used, it was impossible to demonstrate a single *S. pallida*. In one of these cases pieces of tissue from various organs were stained by the Leviditi silver method and the *S. pallida* found present in enormous numbers, although smears from the same organs were negative with the Hastings and Giemsa stains. This would indicate that the organisms present in the dead tissue quickly undergo changes which render them refractory to the ordinary staning methods.

In several cases spirochætæ wert found in sores which did not at all suggest chancres, but proved subsequently to be syphilitic. One patient presented himself a few days after the appearance of two innocent looking ulcers. *S. pallida* were demonstrated in both lesions and the sores healed in less than a week, but it was not until six weeks later that definite evidence of the disease appeared. Vigorous treatment was then begun, but his syphilis was rather stubborn and marked by frequent recurrence of mucous patches. I feel confident that had treatment been instituted when spirochætæ were first demonstrated, the subsequent course of the disease would have been much milder.

Another patient presented himself at the clinic for the treatment of a subacute urethritis and during the examination a small red area, not over 2 mm. in diameter was noted on the glans penis. The area was slightly raised above the surrounding skin, but the epithelium was intact and no induration made out. The patient had not noticed the area. The lesion resembled an ordinary macula and in no way suggested a primary syphilitic lesion. It disappeared entirely in about a week. Large numbers of *S. pallida* were found in the smears and the patient subsequently developed secondaries.

In the condylomata and mucous patches, but particularly the former, the *S. pallida* are usually present in enormous numbers. This corroborates the well-known infectiousness of these lesions.

Being somewhat curious as to whether the *S. pallida* could be demonstrated with any degree of ease in the secondary skin eruptions, a small series of cases was examined. We found them as a rule easily demonstrable in the large well-marked papules, not quite so numerous in the small papules and pustules, and found as a rule only after a long search in the macular lesions.

The results of our investigation show quite conclusively that the *S. pallida* can be found with comparative ease in the pri-

mary sores. We were able to demonstrate them in 90 per cent of our cases. Usually a few minutes was all that was necessary, provided good smears had been obtained, and it was but seldom that a search of more than fifteen minutes was required to find the organism. When one becomes familiar with the morphology of the *S. pallida* and with that of the *S. refringens*, the organism most likely to be confounded with the *S. pallida*, it is seldom that any difficulty is experienced in differentiating between the two.

In our genitourinary clinic smears are made from all venereal sores as a matter of routine and whenever the *S. pallida* is found specific treatment is begun at once. The presence of the *S. pallida* in the lesion is sufficiently positive to warrant the immediate institution of treatment.

THE CONTRAST IN THE EXCRETION OF CHLORINE IN INFLUENZA PNEUMONIA AND IN ORDINARY LOBAR PNEUMONIA.

By LEONARD GEORGE ROWNTREE, M. D.

(From the Medical Clinic of the Johns Hopkins Hospital.)

It has long been known that the excretion of chlorine varies markedly in different diseases. Two diseases, pneumonia and nephritis, have attracted particular attention in this regard. In pneumonia we frequently encounter complete retention, and much discussion has been evoked concerning its prognostic and diagnostic importance in this disease. Formerly it was thought to be of great prognostic value, and is still considered so by some authorities.

A marked retention usually occurs before the crisis in fevers, and immediately after this the excretion is greatly increased. Sahli considers this to be as important an indication of improvement as is the fall in temperature. Achard and Laubry have applied the term "chlorine-crisis" to this condition, but from later work we know that this increase in chlorine may occur before or during the fall in temperature, and often is the first indication of improvement in the pneumonia.

Sahli also attributes great diagnostic value to the excretion of chlorine, for he says: "A pronounced diminution or absence of chlorides in the urine in a febrile disorder will always suggest pneumonia." Certainly pneumonia exhibits a more marked and more constant retention than any other febrile disorder.

Musser and Norris in Osler's Modern Medicine give a very interesting table of the condition of the excretion of chlorides in 436 cases of pneumonia.

	Ashton and Landis.	Musser.	McCrea, Fyshe, Ainley.	Haddon, McKenzie, Ord.	Total.	Per cent.
Chlorides, normal.....	18	6	4	56	84	19.2
Do. decreased...	66	18	9	139	232	53.2
Do. absent	72	20	3	25	120	27.5

A second table of the Haddon, McKenzie, Ord group is given.

	Number of cases.	Number of deaths.	Per cent. of deaths.
Chlorides, absent	25	7	28
Do. diminished	139	22	15.8
Do. normal.....	56	7	12.5

Emerson has studied the records of 34 cases of pneumonia and gives the following interesting report:

Six of these cases had a crisis. The chlorides increased before, during, and after the fall in temperature in different cases.

Twenty-two were with lysis, in fourteen of which there was a drop in the chlorides towards the lysis. On the first day of the lysis in ten cases there was more than 1 gm.—average 2.6. gm. The excretion was lowest during the fall in one-third, and just before the temperature began to fall in two-thirds of the cases.

Five fatal cases showed a steady fall till death, except one which showed slight increase before death. One of these showed a complete retention for six days, when death supervened.

One case of delayed resolution did not show a marked decrease, the excretion varying from 5 to 10 gm. per day for three weeks.

Von Noorden says that only prolonged clinical experience can show whether the appearance of the retention of chlorine has a far-reaching significance as regards prognosis, or whether it is only indicative of a temporary condition. Be this as it may, the fact yet remains that we usually do have a marked retention in pneumonia, and this is generally considered to be of real diagnostic importance.

In health, the normal output of chlorine, estimated as sodium chloride, is 10 to 15 gm. per day, rarely more. The cause of this retention in pneumonia is difficult to explain. It is not due to the decrease in diet, as other diseases on a similar diet exhibit a much greater chlorine output. That it is a true retention has been demonstrated by repeated experiments in which large doses of sodium chloride have been given subcutaneously or by the mouth, without any increase in the excretion of chlorine as occurs in health under similar experiments.

Many theories have been advanced to explain this. Terray ascribes it to the retention of water. This in itself is insufficient.

Röhman says that it is due to the fact that the organ protein-poor in chloride becomes converted in the circulating fluid into protein-rich in chloride.

Von Limbeck, Schwarz and Hijmans claim that the elimination of chlorine is inversely proportional to that of phosphoric acid, and believe that chlorine retention occurs only when the osmotic equilibrium of the body juices has been disturbed, as a result of the increased phosphoric acid elimination. Retention thus preserves the isotonicity of the blood. This does not help us much as the elimination of phosphoric acid varies more than that of chlorine.

A fourth theory has been advanced, which is that in pneumonia the precipitation of albumen in the pulmonary exudate "ties up" large quantities of chlorides. This view is unsatisfactory, for Preble has shown that consolidated areas of lung show an average of 1.69% of sodium chloride, whereas normal lung shows 1.4%. The difference is too slight to account for the phenomenon. But while the proportion by weight is only slightly increased yet the total weight of the lobes involved is greatly increased, so that this will account for considerable retention. But the degree of retention does not bear any direct relation to the amount of consolidation, which also seems to disprove this theory.

Aufrecht believes that it is due to the fact that in fevers the products of catabolism are taken up by the circulation, but only a part of them are immediately transferred into their final products, the remainder being stored in the body by combining with the sodium chloride in the blood plasma.

Though these many theories are advanced, we are not yet certain of the real cause of the retention.

The chlorides of the saliva are relatively greatly increased, but the total daily output by this means is so small that it does not account for the diminution of chlorides in the urine.

The patients on whom the following observations were made were all suffering from lobar pneumonia clinically. In some of them the bacteriological examination of the sputum revealed the *B. influenzæ*. Cultures in these cases showed the *B. influenzæ* in pure or mixed growth, and when mixed cultures were obtained the *B. influenzæ* was the preponderating organism. During this time there was an epidemic of influenza in Baltimore. So in these cases we have assumed the *B. influenzæ* to be the probable etiological factor, and henceforth only refer to the condition as influenza pneumonia.

From a pathological point of view these cases are most frequently of the broncho-pneumonic type, but the process is often limited to one, two or three lobes; and macroscopically on superficial examination it often closely resembles certain stages of croupous pneumonia by the coalescing of contiguous areas of involved lung. Microscopically the exudate is strikingly rich in cellular elements and the fibrin is much less abundant than in croupous pneumonia.

The following observations were carried on in the Clinical Laboratory of the Johns Hopkins Hospital at the suggestion of Dr. Barker, who thought that the excretion of chlorine might differ in these two types of pneumonia.[1]

Total Output of Chlorine in Twenty-four Hours.—In lobar pneumonia this was very low; most of the readings were below 1 gm. Only two cases went above it; one only on the last reading, while the other case was higher throughout. Only two readings were above 3 gm. One patient had absolute retention, starting on the 14th of the month; on this day 10 gm. of sodium chloride were administered but retention continued absolute till the 18th, on which day 2 gm. were excreted. Patient died on the 19th. So here we see a marked retention throughout all our cases, and only on two readings did the output reach as high as the majority of low readings in the influenza pneumonia.

In influenza pneumonia the decrease was not nearly so marked. Only one reading was below 1 gm. Two cases showed a fair amount of retention—3 gm., and the one above referred to showed a marked retention on first reading. All three quickly rose to 5 gm. Then there was a more or less steady increase with increasing diet until an approximately normal daily output was reached. The other two cases were very different. One had a normal output throughout while the temperature was 103° F. When the temperature reached normal, the chloride output was greater than in health. At this point diarrhœa started and its effect on chloride excretion is nicely displayed, *e. g.*, a very marked fall in total chloride output, with a marked increase of chlorine in 10 ccm. of urine, and a marked diminution in the quantity of urine. The other case probably had some tubercular infection along with the influenza pneumonia. The output throughout was fair and reached normal at the time the patient was discharged.

The Amount of Chlorine Excreted in 10 ccm. Urine.—Taking 1400 ccm. as the normal daily quantity of urine and 12 gm. as the average normal daily sodium chloride output, the normal amount of $\frac{N}{10}$ $AgNO_3$ solution necessary to precipitate the chlorine in 10 ccm. of urine is 14 or 15 ccm. We have expressed the amount of chlorine in 10 ccm. of urine in terms of the number of cubic centimeters of $AgNO_3$ solution used because it is more easily charted as such. The number of cubic centimeters of $AgNO_3$ solution multiplied by .005837 gm. gives the amount of sodium chloride in 10 ccm. of urine.

In lobar pneumonia the number of cubic centimeters of $\frac{N}{10}$

[1] The method of work was as follows: All those cases of pneumonia coming into the hospital in which the micro-organism etiologically concerned was undetermined were observed. After the organism was found to be other than the *B. influenzæ*, observations were *discontinued*, as we are already familiar with the excretion of chlorides in pneumonia due to the *B. pneumoniæ*.

The total daily quantity of urine was secured and the chlorides estimated. In all these estimations the Lütke Martins method was used and the results charted.

●——●——● represents the total output of chlorides for 24 hours.

□——□——□ represents the number of ccm. of $\frac{N}{10}$ $AgNO_3$ solution necessary to precipitate the chlorine in 10 ccm. of urine.

●—·—●—·—● represents the total daily output of urine.

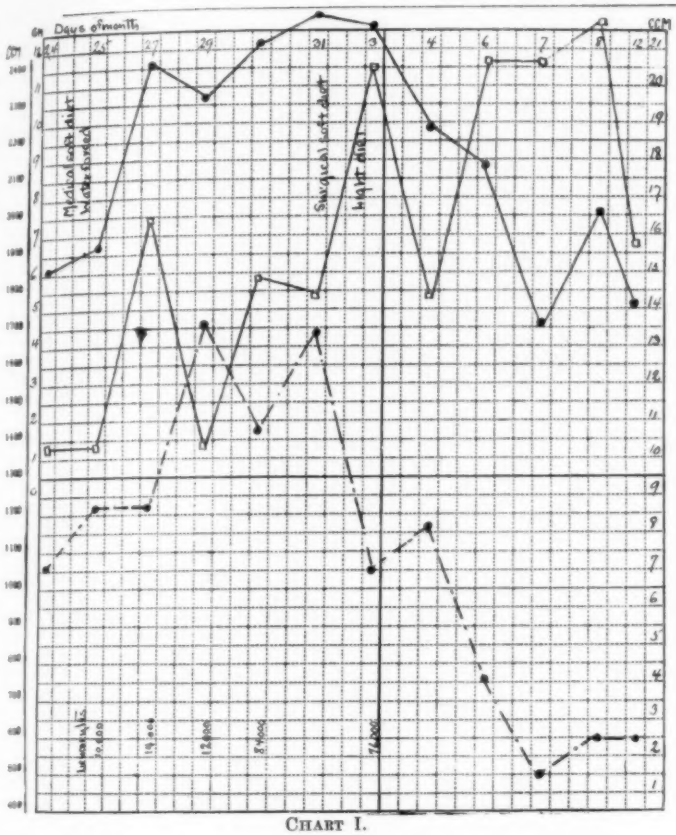


CHART I.

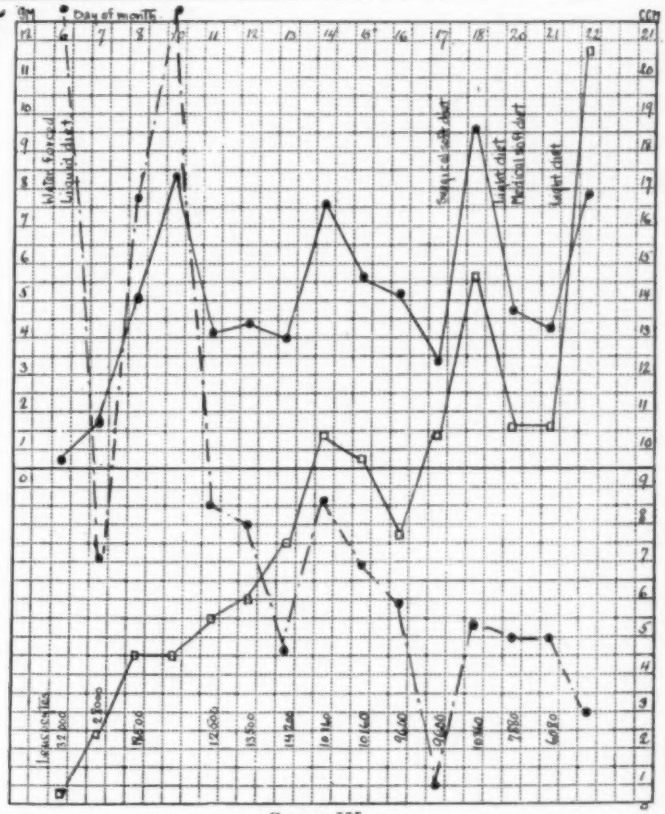


CHART III.

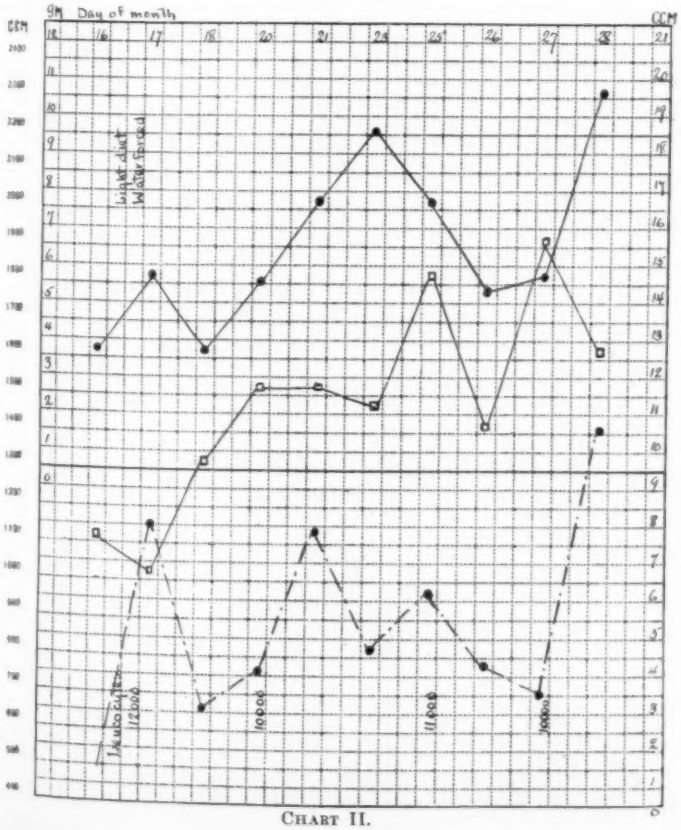


CHART II.

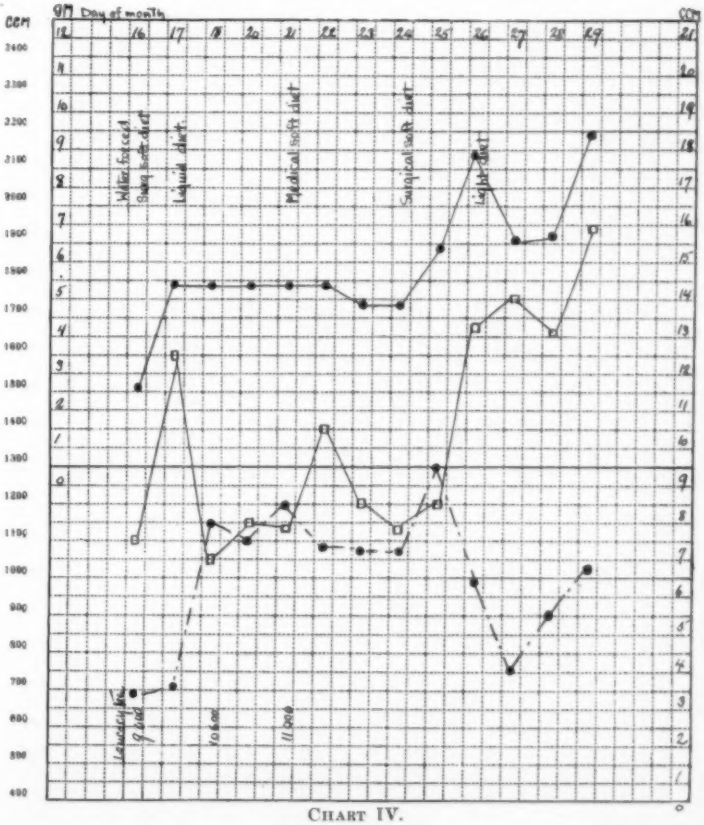


CHART IV.

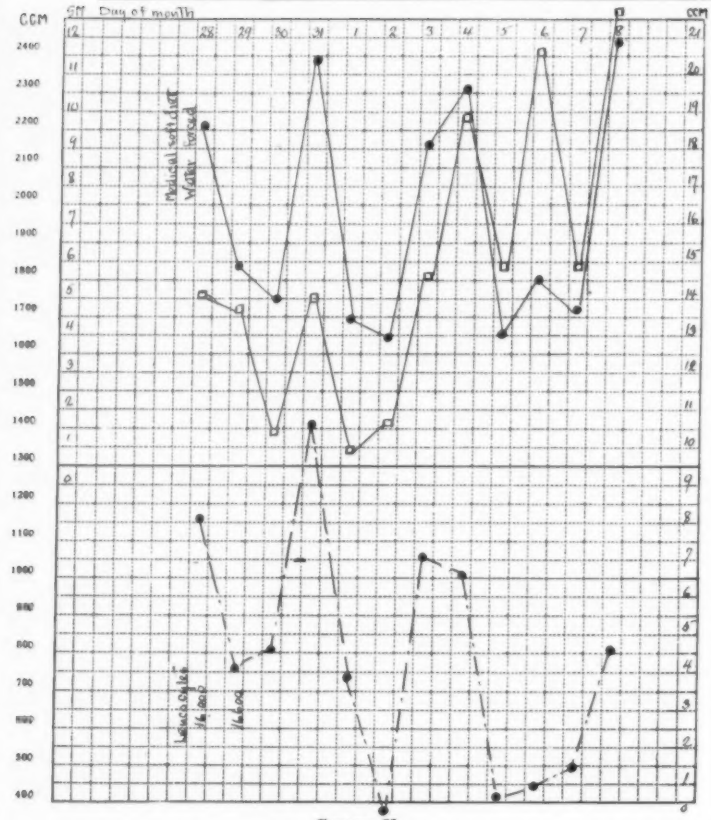


CHART V.

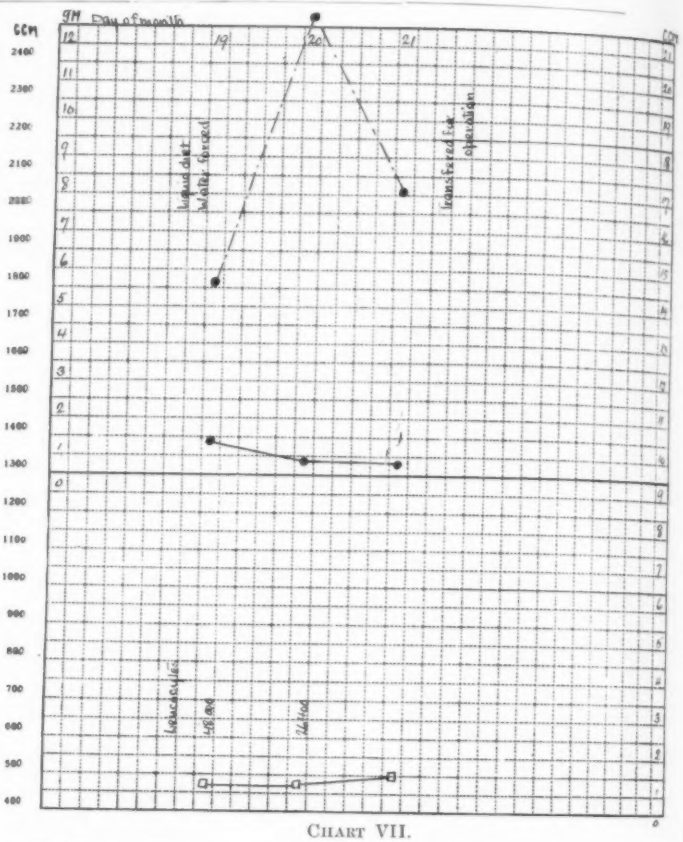


CHART VII.

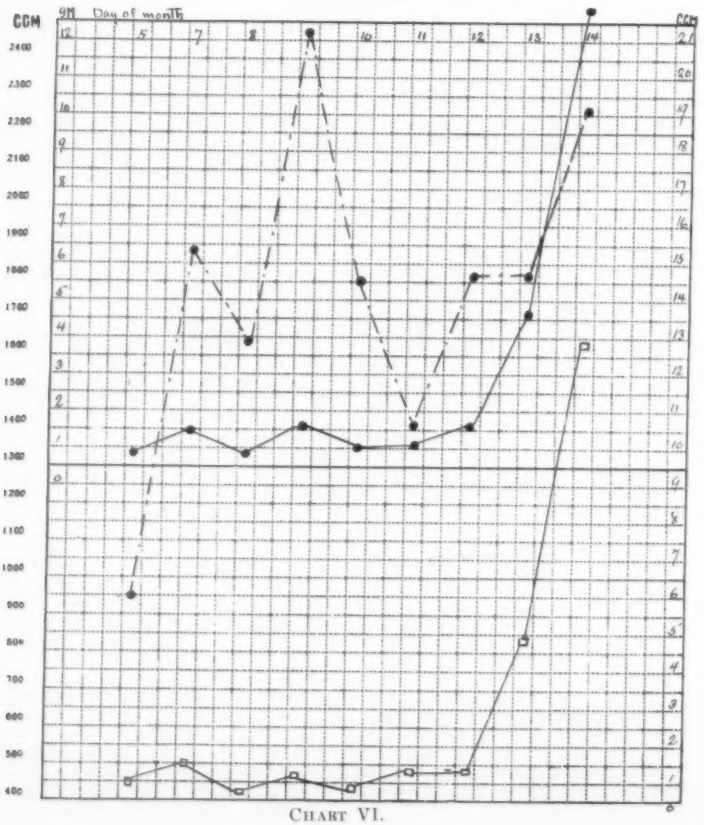


CHART VI.

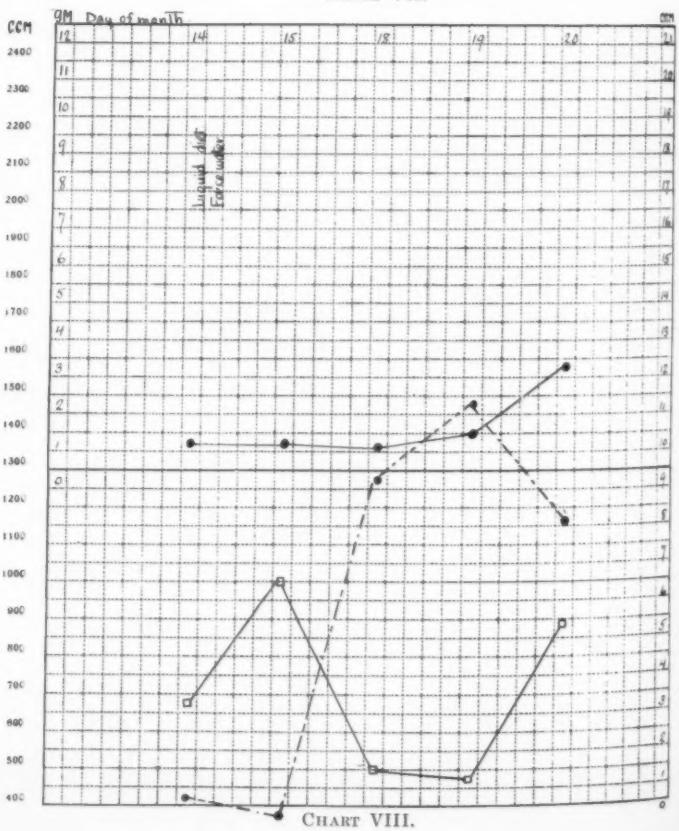


CHART VIII.

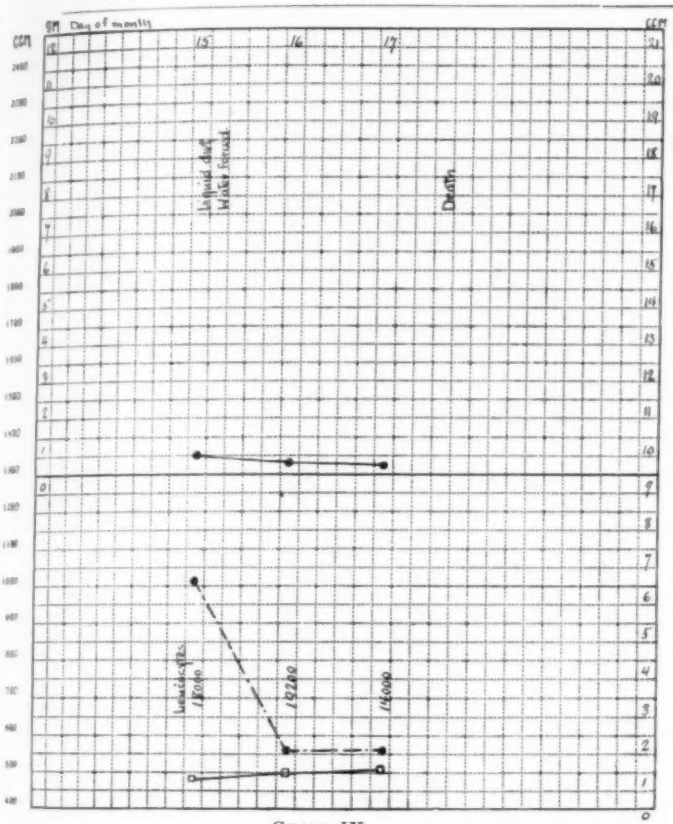


CHART IX.

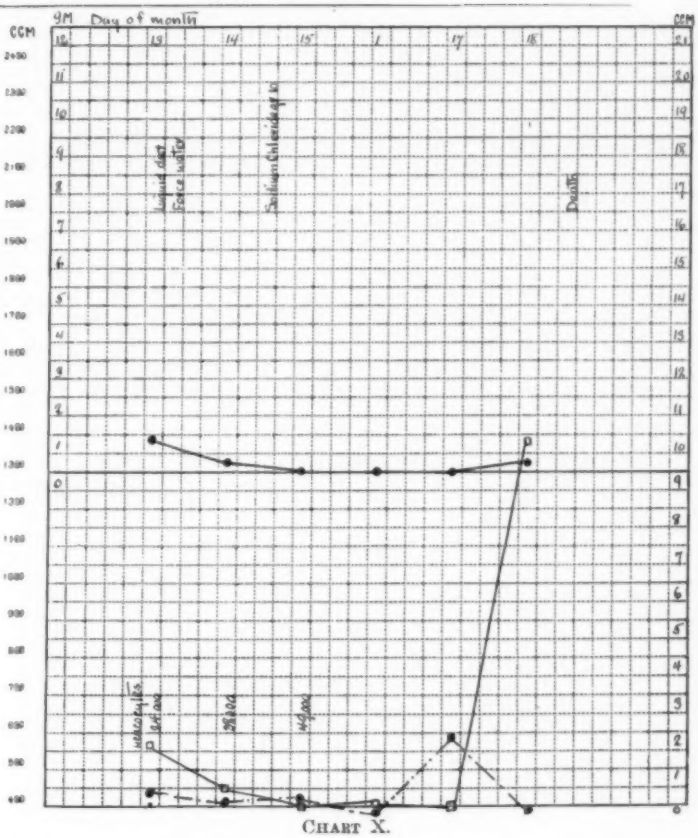


CHART X.

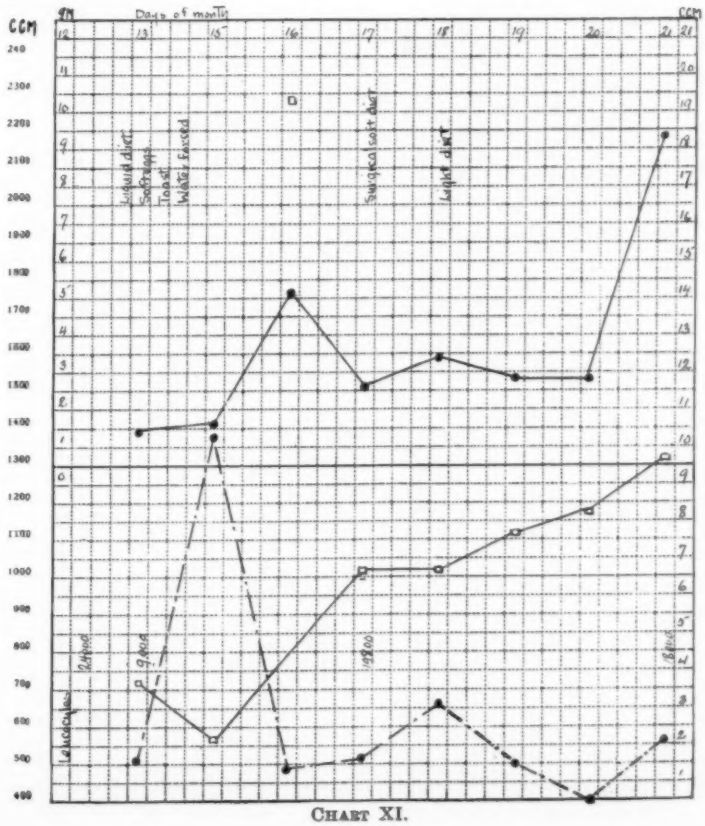


CHART XI.

$AgNO_3$ solution necessary to precipitate the chlorine in 10 ccm. of urine was low. In only four instances did it exceed 7 ccm.; and of these four, three readings were in the same case. One case registered zero on three successive days. The most peculiar chart is that of W. (Chart XI). The sudden rise to 19 ccm. was associated with no condition which would explain the rise. It quickly dropped and then showed a steady rise until 9 ccm. was reached.

Influenza pneumonia, on the other hand, did not display a low amount of chlorine to 10 ccm. of urine. Four cases started at 7 ccm. or above it, while the fifth started near zero. The elimination, if estimated as the amount of chlorine in 10 ccm. of urine, is very similar in all. One feature is common to all these cases as each patient exhibited a more or less steady rise until the normal point or one above it was reached. In each case the reading went above normal at the close of the observations. So, early in influenza pneumonia we see a fair amount of chlorine in 10 ccm. of urine, with a gradual increase to normal.

Daily Output of Urine.—In lobar pneumonia the output was low except in one case in which it varied from 1800 to 3000 ccm. The average daily output was less than a liter. The only constant feature observed was that in each instance when the daily output of urine was large the amount of chlorine in 10 ccm. of urine was very small.

In influenza pneumonia the quantity of urine was very variable. Two started with small quantities, two fair quantities, and one a large quantity. All the cases passed a normal quantity during some part of the course. All exhibited a tendency to decrease in quantity as the patient improved, and associated constantly with this was an increase in the amount of chlorine in 10 ccm. of urine. A rise in the quantity of urine was not necessarily associated with a decreased amount of chlorine in 10 ccm. of urine, as was observed in lobar pneumonia. The average daily output was fair throughout but was somewhat low towards the end.

Leucocytes.—All the cases of both types exhibited leucocytosis with no marked contrast. But as a rule the leucocytosis was higher and more constant in lobar pneumonia. In influenza pneumonia the gradual decrease of leucocytes was associated with a diminution in the quantity of urine and increase in the amount of chlorine excreted in 10 ccm. of urine.

Temperature.—We wish to state that these cases of influenza pneumonia were not admitted at the onset of the disease. There was no definite onset in these cases as in lobar pneumonia, but merely a general feeling of ill-health for a time varying from a few days to three weeks. Three of our lobar pneumonia cases were observed late in the disease. In all of our cases the patients were very sick when observations were started and the diagnosis of the cause was not apparent, so that we feel that we are justified in contrasting the chloride excretion in the two series.

The temperature declined gradually by lysis in all the influenza pneumonia cases and was associated with gradual fall of leucocytes, diminution in the quantity of urine, and increase in the amount of chlorine excreted in 10 ccm. of urine.

Blood pressure was low in the cases observed, three cases of both series going below 90 mm. systolic pressure.

Conclusions.

1. That the retention of chlorine did not exist in the cases of influenza pneumonia studied to nearly so marked a degree as is usually found in acute lobar pneumonia.

2. That in uncomplicated cases of influenza pneumonia there may be a normal daily excretion of chlorine; that if it is reduced, the reduction is not very great and does not remain low, but steadily increases by lysis till normal is reached.

3. That there is a tendency for the amount of chlorine in 10 ccm. of urine and the total daily output of chlorine to run more or less parallel in their fluctuations.

4. That the quantity of urine during the course of influenza pneumonia is not greatly diminished, and that the amount of chlorine in 10 ccm. of urine is not markedly low; that the urine has a slight tendency to decrease in quantity during convalescence, while the amount of chlorine in 10 ccm. of urine steadily increases to normal.

5. That in influenza pneumonia a large quantity of urine is not usually associated with a markedly low amount of chlorine in 10 ccm. of urine, as is usually the case in ordinary lobar pneumonia.

In concluding I wish to thank Dr. C. P. Emerson and Dr. T. R. Boggs for their suggestions and help in carrying on this work.

NOTES EXPLANATORY OF CHARTS.

I. C., male, white, æt. 32. Influenza pneumonia. Five weeks before admission patient had a chill followed by fever. He went back to work, but could not keep it up. Had to stop 2 weeks on account of cough, which grew worse. Pain developed later. Entered hospital January 20. Temperature on 24, 103°; on 27, 101°. Left lower lobe involved. *B. influenzæ* in culture from sputum on 26. Diarrhœa began February 3 (4-12 stools a day). Blood pressure, systolic, 118 to 98 mm.

II. V., male, white, æt. 30. Influenza pneumonia. Rather gradual onset following debauch about Christmas. Temperature 100.5° on 16 and 17; reached normal on 20. Right lower lobe involved. January 24, *B. influenzæ* found in culture from sputum. Blood pressure 102 mm.

III. J. W., negro, æt. 23. Influenza pneumonia. Onset on December 28, with pain in the side; cough for a week previous to this. Right lower and middle lobes involved. Blood pressure on January 5, 105 mm.; on 8, 100 mm.; on 14, 78 mm. Patient recovered and was transferred to surgical side for an operation on his femur.

IV. C., male, white, æt. 65. Influenza pneumonia. Patient had been sick for nearly two weeks. Temperature 102.6° on January 16; normal on 26. Right upper and lower lobes involved. Blood pressure 85 mm. on admission; 110 mm. on 26.

V. T., male, white, æt. 32. Influenza pneumonia and pulmonary tuberculosis. Onset was gradual. Temperature from 104° to 101.5°; reached normal in the mornings after January 28. *B. influenzæ* found in culture from sputum. Left upper lobe involved—case more like one of lobar than broncho-pneumonia (Dr. Emerson). Blood pressure 88 mm. *B. tuberculosis* found in sputum February 6.

VI. C. M., male, white, æt. 24. Acute lobar pneumonia. Chart shows chlorine crisis. Right upper, middle and lower lobes involved; and left lower (?). Temperature was normal for first time on January 12.

VII. R., negro, æt. 28. Lobar pneumonia and empyema. Onset with chill February 10; pain in side February 14. Temperature 104.4°. Left lower lobe involved. Blood pressure 80 to 88 mm.

VIII. C., male, white, æt. 19. Acute lobar pneumonia. Onset February 5 with pain in side, cough, fever, and rusty sputum. Right and left lower lobes involved. Temperature 99.5° on February 14; normal on February 18. Blood pressure 118 to 100 mm. Discharged well on February 26.

IX. F., negro, æt. 24. Lobar pneumonia. Onset sudden. Temperature 104° on January 15; 100.5° on 17. Both lower lobes involved. Blood pressure 88 to 75 mm.

X. R., negro, æt. 19. Acute lobar pneumonia. Onset sudden March 18 with chill, pain in side and fever. Right upper, middle, and lower and left-lower lobes involved. Temperature 102° until death. Blood pressure 75 to 100 mm.

XI. W., male, white, æt. 18. Acute lobar pneumonia. Onset February 6 with chill, pain in the side, and fever. Right middle and lower lobes involved. Temperature above 100° on February 13; normal on February 17. Blood pressure 100 mm. Discharged well on February 29.

ERRATA.

JOHNS HOPKINS HOSPITAL BULLETIN, NOVEMBER, 1908.

Page 337, column I, line 3, for "which," read "but."

Page 338, column II, line 5, for "sufficing," read "sufficed."

NOTES ON NEW BOOKS.

Cholécysto-Pancréatite: Essai de Pathogénie. Par LE DOCTEUR REINE MAUGERET. (*Paris: G. Steinheil, 1908.*)

This monograph is one of rather more than usual importance, for it presents a new—or at least an unorthodox—theory of the pathogenesis of pancreatitis, and not only pleads for it on the basis of experimental work, but also fortifies the position taken, by a closely-reasoned consideration of the clinical phenomena of the disease.

The author's thesis (it was first really advanced by his teacher, M. Thiroloix, in a clinical lecture at the Saint Antoine Hospital in 1907), and the conclusions reached may first be set down; the reasoning which led up to them will then be reviewed in some detail.

The excellent introductory historical chapter of Maugeret's monograph shows that, though various channels of infection in pancreatitis had been suggested by Klippel as early as 1897 (ascending infection from the intestine through the bile ducts, ascending infection *via* the blood stream, infection by contiguity, and infection *via* the lymphatics) the practically unanimous opinion, at least, of surgeons, had been that pancreatitis was the result of an infection starting from the duodenum and reaching the organ by the duct of Wirsung. Tuberculous pancreatitis was regarded as an exception, after its lymphatic transmission had been established by Klippel; the ordinary infections were always assumed to pass up the ducts; an obstruction of the duct of Wirsung (as Mayo Robson held) being the principal determining cause of the condition and gall-stones being the usual form of obstruction. Desjardins (1905) regarded pancreatitis and cholelithiasis as contemporaneous phenomena—both due to intestinal infection which, in the gall-passages, produces stones, in the pancreas, inflammation.

The theory of M. Thiroloix, on the other hand, is as follows: Every cholelithiasis begins as a mild cholecystitis, which may and often does lead to a deposit of cholesterin and the formation of calculi. This infection of the gall-bladder tends to chronicity, and becomes a constant source of danger to neighboring organs. It leads to localized peritonitis over duodenum, pylorus, colon, and gall-bladder; it leads to localized hepatitis; but, most important of all it travels up the lymphatics and leads to the sclerotic changes in the pancreas so long recognized as a frequent concomitant of gall-stones. "In the genesis of biliary pancreatitis the infection and not the calculus is the important factor." To test the validity of this assumption Maugeret undertook his clinical and experimental research which led him to the following conclusions: 1. Pancreatitis co-existing with cholelithiasis is neither an independent nor a contemporaneous and parallel affection due to the same initial cause. It is a true complication. 2. Its starting point is neither directly nor indirectly an intestinal infection. 3. The infection does not reach the pancreas by the duct. 4. The infection does not reach the pancreas by contiguity. 5. In all cases of cholelithiasis, no matter what their nature (*i. e.*, no matter what the infecting agent?) the cause of the pancreatitis is the same. 6. This sole cause of pancreatitis in cholelithiasis is the infection of the gall-bladder. 7. The gall-bladder infection reaches the pancreas by the efferent vessels of the gall-bladder which empty into a group of glands near the head of the pancreas, the same group receiving lymphatics from the gland itself. 8. Infection of the pancreas *via* the lymphatics is not only possible, but frequent.

The argument. 1. Anatomical basis. Briefly, the facts are these. The lymphatics of the pancreas and those of the biliary tracts all converge toward the same spot—namely, the head of the pancreas; and here there is free anastomosis among them. The head of the pancreas is, therefore, a "carrefour lymphatique"; into it empty the lymphatics of the gall-bladder, the biliary tracts, the liver, and the pancreas itself. These facts fit in well with Thiroloix's hypothesis as to the genesis of pancreatitis; and they explain fully the elective predominance which pancreatic lesions show for the *head* of the organ. (But one hesitates to go along with Maugeret and speak confidently of a cholecysto-appendicitis, analogous to this cholecysto-pancreatitis, the infection reaching the vermiform appendix by the mesenteric lymphatics. At least, such a clinical entity is, as yet, a conception, as Maugeret would say "assez vague." The suggestion is, however, interesting; no doubt, lymphatic transmission of infection through the abdomen has not received sufficient attention.)

2. Pathological considerations. "Enlarged glands in the small omentum," says Mayo Robson, "may be found in pancreatitis as well as in cancer." Kehr, on the other hand, makes their absence in pancreatitis a point in the differential diagnosis from carcinoma. They are certainly present in tuberculous pancreatitis, and on their presence Klippel based his theory of the lymphatic transmission of this disease. Enlarged glands have been frequently reported; but they are not constantly seen, being overlooked, says Maugeret, on account of their softness. (The argument here is weak; if the glands are so frequently overlooked, how do we know that they are usually present? And might not their presence, in any case, be a *result* and not a mediate *cause* of the pancreatitis?)

Pancreatitis is most often associated with stone in the common duct. This does not mean that the common duct stone has caused the pancreatitis, but that the pancreatitis (originating in the gall-bladder infection) has caused the calculus to lodge in the common duct, which—partially blocked by œdema and swelling

of the pancreas—refuses to allow the gall-bladder stone to pass through its intra-glandular portion. One, therefore, often sees pancreatitis without stone in the common duct; but never stone in the common duct without pancreatitis" (! and also ?).

3. The possibility of ascending infection through the duct. This, though always assumed, has never been demonstrated. Only two proven instances of ascending infection are known; the first was in the experiment of Carnot, who brought it about by placing a thread in the duct of Wirsung and thus produced wholly artificial conditions; and the second instance is in the rare cases of pancreatitis associated with lumbricoid worms in the duct, an entirely exceptional condition. The well-known observation of Opie and Halsted is also regarded by Maugeret as representing quite an unusual state of affairs. Even if biliary reflux *did* take place frequently, pancreatitis would not be thereby explained; for Claude Bernard observed cases of such a spontaneous reflux in man (as evidenced by staining of the mucosa) without lesions in the pancreas. ("But," one must ask, "was the bile infected in these cases?")

Furthermore, the pancreatic juice is bactericidal; its current is constantly efferent; and the normal ærobic and anærobic flora, which Gilbert and Lippmann have shown to exist within the distal 2 or 3 cm. of the duct of Wirsung, are either swept away or killed. These facts speak against the possibility of ascending infection. (This chapter raises many questions. On the whole, the logic is sound; it is easy to attack it at many points, but space forbids it here. One regrets that the problem of ascending infection of the kidney from the bladder does not seem to have occurred to the author as analogous with his own. This problem has been argued about for years; a consideration of the points brought forward in the debate would have been very useful in providing analogies; the conclusions reached would have supported Maugeret's contention, for there is little doubt that infection, though by no means rarely reaching the kidney *via* the ureter very often takes another path.)

4. Therapeutic considerations. The treatment of pancreatitis is essentially surgical and consists in drainage, *i. e.* disinfection of the gall-bladder. Simple removal of the obstacle (the common-duct stone) is not enough; it is the disinfection which does the work, and which "is always efficacious." This is the strongest argument against the intestinal origin of pancreatitis and in favor of the idea that the infection originates in the gall-bladder. Cholecystectomy, rather than simple cholecystostomy, is suggested as the more radical procedure in the treatment of pancreatitis; and the opinion of Lejars and Quénu is quoted with approval that "in gall-stones of the gall-bladder, simple cholecystectomy without drainage may be, and indeed in many cases has been shown to be, sufficient."

(Maugeret quotes three clinical cases to support his contention. In two, simple choledochotomy with removal of the stone was done, and in both of these the pancreatitis persisted; in the third cholecystectomy, with drainage of the hepatic duct, was performed and in this patient the pancreatitis was cured. One must remark, however, that simple choledochotomy without drainage of the gall-bladder is insufficient treatment for pancreatitis *no matter how the infection reaches the pancreas*. Furthermore, one cannot accept off-hand the statement that the "pancreatitis persisted," in view of the great difficulty experienced in making a diagnosis of this condition, even after study of the fat-content of the stools; though it would not be at all surprising if it *did* persist after such inadequate treatment. In any event the number of cases reported is too small to warrant conclusions. One hesitates, too, to accept Maugeret's suggestion that cholecystectomy be substituted for cholecystostomy on the principle, apparently, that if drainage is good ablation must be better. Lejars and Quénu are quoted as saying that "in simple gall-stones of the gall-bladder, cholecystectomy without drainage may be sufficient"; which runs somewhat counter to orthodox surgical teachings; though they save themselves, to an extent, by conceding that drainage, which is often a useful complement, "is particularly indicated in infected lithiasis.")

5. Experimental basis. Dogs were the animals used and nine experiments were done. The gall-bladder was injected with .5 to 3 cm. formol and the dogs killed from 2 to 46 days after the injection. Attempt was made to determine the path of absorption by introducing into the gall-bladder, along with the formol, a small amount of powdered charcoal; but the attempt failed. The charcoal was never found except in the gall-bladder or ducts. The formol produced, of course, a chemical cholecystitis; but the important fact was that when the dogs were killed they were found to have either an acute or a chronic pancreatitis. The acute condition was represented in one case by a hemorrhagic pancreatitis; in another by pancreatico-peritoneal hemorrhage with cysto-steato-necrosis. The authors also observed accentuation of the normal lobulation (amounting sometimes to actual "morcellement"); great increase in the number and size of the islands of Langerhans, with the existence of transition forms between them and the acini; intense congestion; in every case edema and leucocytic infiltration. The chronic inflammation took the form of an inter-lobar and inter-acinous sclerosis. The experiments proved, therefore, that pancreatitis could be brought about by producing cholecystitis and also afforded an opportunity to study the histogenesis of the disease—an opportunity of which the author has made good use.

(There is no doubt about the importance and interest attaching to this experimental contribution. The study of such a complex problem raises many questions, which one must leave unanswered if the research is not to be allowed to stray from its original purpose. One regrets, however, that the study of the path of absorption was abandoned without ado because, forsooth, the "charcoal particles were perhaps too large to be absorbed." This was, indeed, the essential question and the answer should have been found at all costs. It is to be regretted, also, that infectious cholecystitis was not produced; this would have reproduced clinical conditions more exactly than a chemical cholecystitis; some easily recognizable organism could have been used and its passage through the lymphatics demonstrated. The absorptive function of the gall-bladder might have been studied; its known capacity for retaining organisms for long periods of time (as after typhoid fever) would seem to indicate its poor absorptive power, and this *may* have been why charcoal was never found in the lymphatics. If the gall-bladder is at all analogous to the urinary bladder one would expect it to do very little absorbing. Another noticeable lack is the absence of good notes about the histological changes in the common duct; there is little more in the protocols than a report of macroscopic findings or the entirely trivial announcement that the "cholédoque contient une boue charbonneuse.") J. C.

Cancer of the Womb: Its Symptoms, Diagnosis, Prognosis, and Treatment. By FREDERICK JOHN MCCANN, M. D., F. R. C. S., M. R. C. P. 172 pages. 20*s.* net. (*London: Oxford University Press, 1907.*)

In a monograph of 172 pages the author presents the results of his wide personal experience and observation concerning carcinoma of the uterus. After a concise description of the normal anatomy and physiology of the uterus, the etiology of cancer in general, and especially of uterine cancer, is briefly considered. In the following chapters the pathology, clinical history, diagnosis, prognosis, and treatment of the various forms of uterine carcinoma are discussed. Detailed descriptions of cases representing the different varieties of cancer are given and are illustrated by reproduction of photographs and photomicrographs. I cannot

agree with the author that photographs give a truer representation of the appearance of the specimen than reproduction of good drawings. For the most part the photographs are hazy, and the distinction between normal and pathological tissues is ill-defined. The failure of photography to do more than give mere outlines in the case of anatomical specimens, is probably often due to the presence of blood and moisture on the surface; whereas in making a drawing of the tissue the artist is not influenced by adventitious conditions.

The author rightly condemns the publication in the lay press of descriptions of the disease, and believes that more good can be accomplished by the better education of the general practitioner and the instruction of mid-wives and district nurses, so as to enable them to warn their patients to seek a consultation on the earliest appearance of any abnormality. The early diagnosis by means of the microscope is strongly urged. The author's advocacy of supravaginal amputation of the cervix in some cases of early carcinoma cannot be too strongly condemned. The only hope of increasing the appallingly low percentage of permanent cures in carcinoma of the cervix rests in the radical extirpation of the uterus and parametrium in early stages of the disease. As Dr. McCann states that the recurrences in these cases were almost without exception not at the site of operation, but in the parametrium, the better prognosis of a radical operation is evident.

Sarcoma of the uterus is briefly considered, and this is followed by a chapter on deciduoma malignum. The short chapter on postoperative treatment is excellent, and if the advice contained in it were more generally followed, many patients would be benefited.

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